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TRANSACTIONS

OF THE

PATHOLOGICAL SOCIETY

OF PHILADELPHIA.

VOLUME XVI.

CONTAINING THE REPORT OF THE PROCEEDINGS FROM SEPTEMBER, 1891, TO JULY, 1893.

EDITED BY

ALFRED STENGEL, M.D.,

INSTRUCTOR IN CLINICAL MEDICINE IN THE UNIVERSITY OF PENNSYLVANIA; PATHOLOGIST TO THE GERMAN HOSPITAL; MEDICAL REGISTRAR TO THE PHILADELPHIA HOSPITAL; RECORDER OF THE SOCIETY.

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1893.

ERRATUM.

Page 100. "Dr. G. W. Ziegler" *should read:* Dr. Jacob L. Ziegler.

CONTENTS.

	PAGE
List of Former Presidents	iv
List of Officers and Committees elected 1891	v
List of Officers and Committees elected 1892	vi
List of Members	vii
List of Corresponding Members	xvii

Diseases, etc., of the Osseous System	1
Diseases, etc., of the Digestive System	19
Diseases, etc., of the Vascular and Hæmatopoietic Systems	79
Diseases, etc., of the Organs of Respiration	152
Diseases, etc., of the Genito-urinary System	160
Diseases, etc., of the Nervous System	213
Tumors not otherwise Classified	226
Miscellaneous	232

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FORMER PRESIDENTS.

SAMUEL D. GROSS, M.D., LL.D., D.C.L. Oxon., LL.D. Cantab., elected 1857.

RENÉ LA ROCHE, M.D., elected 1858.

ALFRED STILLÉ, M.D., elected 1859, '61, and '62.

EDWARD HARTSHORNE, M.D., elected 1860 and '63.

J. M. DA COSTA, M.D., elected 1864, '65, and '66.

JOHN H. PACKARD, M.D., elected 1867 and '68.

S. WEIR MITCHELL, M.D., elected 1869.

JOHN ASHHURST, Jr., M.D., elected 1870.

JAMES H. HUTCHINSON, M.D., elected 1871 and '72.

WILLIAM PEPPER, M.D., elected 1873.

H. LENOX HODGE, M.D., elected 1876.

S. W. GROSS, M.D., elected 1879.

JAMES TYSON, M.D., elected 1882 and '83.

E. O. SHAKESPEARE, M.D., elected 1884.

J. C. WILSON, M.D., elected 1885 and '86.

F. P. HENRY, M.D., elected 1887 and '88.

HENRY F. FORMAD, M.D., elected 1889 and '90.

ARTHUR V. MEIGS, M.D., elected 1891 and '92.

OFFICERS AND COMMITTEES

OF THE

PATHOLOGICAL SOCIETY OF PHILADELPHIA.

(Elected at the annual meeting, October, 1891.)

President.

ARTHUR V. MEIGS, M.D.

Vice-Presidents.

H. R. WHARTON, M.D.,
JOHN GUITÉRAS, M.D.,
J. B. DEAVER, M.D.,
G. G. DAVIS, M.D.

Secretary.

FREDERICK A. PACKARD, M.D.

Recorder.

JOHN L. HATCH, M.D.
(Resigned January 28, 1892.)

ALFRED STENGEL, M.D.
(Elected January 28, 1892.)

Treasurer.

T. S. WESTCOTT, M.D.

Curator.

ALFRED STENGEL, M.D.

Committee on Publication.

G. E. De SCHWEINITZ, M.D.,
H. A. HARE, M.D.,
JAMES TYSON, M.D.,
F. P. HENRY, M.D.

Committee on Morbid Growths.

ARTHUR V. MEIGS, M.D.,
F. X. DERCUM, M.D.,
J. L. HATCH, M.D.,
W. M. L. COPLIN, M.D.,
ALFRED STENGEL, M.D.,
E. L. VANSANT, M.D.,
E. LAPLACE, M.D.

Business Committee.

J. B. DEAVER, M.D.,
H. R. WHARTON, M.D.,
J. H. MUSSER, M.D.,
H. F. FORMAD, M.D.,
F. A. PACKARD, M.D.,
C. W. BURR, M.D.

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OF THE

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J. H. MUSSER, M.D.,
H. R. WHARTON, M.D.,
JOHN B. DEAVER, M.D.,
G. G. DAVIS, M.D.

Secretary.

CHARLES W. BURR, M.D.

Recorder.

ALFRED STENGEL, M.D.

Treasurer.

T. S. WESTCOTT, M.D.

Curator.

CARL HAMANN, M.D.

Committee on Publication.

F. P. HENRY, M.D.,
JAMES TYSON, M.D.,
F. A. PACKARD, M.D.,
J. P. C. GRIFFITH, M.D.

Committee on Morbid Growths.

C. W. BURR, M.D.,
W. M. L. COPLIN, M.D.,
E. LAPLACE, M.D.,
E. L. VANSANT, M.D.,
M. V. BALL, M.D.,
ALFRED STENGEL, M.D.,
F. X. DERCUM, M.D.

Business Committee.

J. H. MUSSER, M.D.,
F. P. HENRY, M.D.,
J. B. DEAVER, M.D.,
F. A. PACKARD, M.D.,
H. R. WHARTON, M.D.

LIST OF MEMBERS.

Members marked O. M. are those originating the Society.
Members marked N. R. are non-resident.

ELECTED

1890 Adams, J. Howe.
1857 Agnew, D. Hayes, died in 1891. (O. M.)
1887 Allyn, Herman B., 310 North Fortieth Street.
1889 Anders, J. M., 1637 North Broad Street.
1867 Ashhurst, Francis, Mount Holly, New Jersey. (N. R.)
1861 Ashhurst, John, Jr., 2000 DeLancey Place.
1891 Ashhurst, W. W. (N. R.)
1891 Ashton, Thomas G., 222 South Eighth Street.

1887 Baker, G. F., 1818 Spruce Street.
1887 Baldy, J. M., 1722 Chestnut Street.
1891 Ball, M. V., Eastern Penitentiary.
1888 Balliet, Tilghman M., 3705 Powelton Avenue.
1880 Bartholow, Roberts, 1525 Locust Street.
1868 Bartles, Wm. H., Insane Department Pennsylvania Hospital, West Philadelphia. (N. R.)
1883 Barton, Isaac, 111 North Sixteenth Street.
1869 Barton, J. M., 1344 Spruce Street.
1885 Beates, Henry, Jr., 1421 Girard Avenue.
1873 Beecher, A. C. W., died 1893.
1871 Bennett, Wm. H., 332 South Fifteenth Street.
1863 Benton, C. H. (N. R.)
1887 Berens, Bernard, 2006 Arch Street.
1870 Bertholet, Robert Morris, died 1882.
1871 Betts, Thomas, Branchtown, Pa. (N. R.)
1892 Birney, D. B., 1810 DeLancey Place.
1860 Bishop, C. S., dead.
1866 Black, J. J., New Castle, Delaware. (N. R.)
1863 Boardman, C. H., St. Paul, Minn. (N. R.)
1893 Bochroch, Max H., 968 North Eighth Street.

ELECTED

1886 Bodamer, George A., 1219 North Fifteenth Street.
1892 Boger, John A., 2213 North Broad Street.
1857 Boker, Charles S., 212 South Fifteenth Street. (O. M.)
1868 Bolles, Lucius S., died August 15, 1873.
1893 Boyer, H. P., Orthopædic Hospital.
1883 Breinig, P. B., Bethlehem, Pa. (N. R.)
1890 Bricker, William H., 227 North Twenty-second Street.
1857 Brinton, John H., 1423 Spruce Street. (O. M.)
1890 Brooke, Benjamin. (N. R.) U. S. Army.
1874 Bruen, E. T., died March 31, 1889.
1893 Bryan, J. Roberts, 4200 Chestnut Street.
1887 Burr, Charles W., 1330 Spruce Street.

1893 Carpenter, H. B., 1205 Locust Street.
1893 Carter, W. S., 807 North Forty-first Street.
1872 Cathcart, James H., died March 5, 1874.
1879 Cathcart, T. H., died March 28, 1882.
1887 Cattell, H. W., 3455 Woodland Avenue.
1890 Cerna, David, University of Texas. (N. R.)
1871 Cheston, C. Morris, West River, Maryland. (N. R.)
1887 Chrystie, Walter, Bryn Mawr, Pennsylvania.
1866 Cleemann, Richard A., 2135 Spruce Street.
1872 Cohen, J. Solis, 1431 Walnut Street.
1883 Cohen, S. Solis, 219 South Seventeenth Street.
1887 Collins, James, 704 Franklin Street.
1890 Conrad, J. Reed, dead.
1890 Coplin, W. M. L., 310 Catharine Street.
1869 Corbit, William B., Washington, D. C. (N. R.)
1871 Curtin, Roland G., 22 South Eighteenth Street.

1857 Da Costa, J. M., 1700 Walnut Street. (O. M.)
1885 Daland, Judson, 319 South Eighteenth Street.
1859 Darby, J. T., dead.
1876 Darlington, W. L. (N. R.)
1857 Darrach, James, 5021 Green Street, Germantown. (O. M.)
1868 Darach, William, died January 28, 1881.
1890 Davis, C. N., 2107 Chestnut Street.
1882 Davis, G. G., 255 South Sixteenth Street.
1893 Davisson, Alexander H., 2204 Pine Street.

ELECTED

1889 Deaver, H. C., 1629 Oxford Street.
1885 Deaver, J. B., 120 South Eighteenth Street.
1882 Dercum, F. X., 810 North Broad Street.
1882 De Schweinitz, G. E., 1401 Locust Street.
1886 Donges, John W., Camden, N. J.
1888 Downs, Andrew J., 1218 North Eighteenth Street.
1889 Downs, Norton, 5056 Main Street, Germantown.
1877 Drysdale, T. M., 1531 Arch Street.
1866 Duer, Edward L., 1606 Locust Street.
1876 Dulles, C. W., 4101 Walnut Street.
1883 Dunn, T. D., West Chester, Pennsylvania. (N. R.)
1883 Dunmire, G. B., 1116 Arch Street.
1860 Dunton, W. R., 5059 Germantown Avenue, Germantown.
1863 Dyer, Ezra, Pittsburg, Pa. (N. R.)

1882 Edwards, W. A., San Diego, California. (N. R.)
1885 Egbert, J. C., Wayne, Pennsylvania.
1875 Engel, Hugo, forfeited membership by non-payment of dues, February 23, 1882.
1891 Eshner, A. A., 224 South Sixteenth Street.
1876 Eskridge, J. T., Colorado Springs, Colorado. (N. R.)

1893 Farr, W. W., 2112 DeLancey Place.
1892 Fenn, William B., Colorado Springs. (N. R.)
1881 Fenton, T. H., 1335 Arch Street.
1857 Fischer, Emil, 729 North Sixth Street. (O. M.)
1863 Fish, Augustine H., died August 3, 1872.
1876 Fisher, Henry M., 317 South Twelfth Street.
1890 Flick, L. J., 736 Pine Street.
1869 Ford, William H., 1622 Summer Street.
1878 Formad, H. F., died June 5, 1892.
1888 Fox, J. M., Torresdale. (N. R.)
1886 Freeman, W. J., 2034 Chestnut Street.
1889 Frese, Carl, German Hospital.
1880 French, M. S., 1437 Spruce Street.
1890 Friebis, George, 1720 Chestnut Street.
1885 Fussell, M. H., 169 Green Lane, Manayunk.

1883 Garrett, E. F., died 1891.
1871 Gerhard, George S., Ardmore, Pennsylvania. (N. R.)
1870 Getchell, Frank H., 1432 Spruce Street.

ELECTED

1890 Gibb, Joseph S., 817 Franklin Street.
1877 Gilliard, Louis, died November, 1878.
1890 Gleason, E. Baldwin, 41 South Nineteenth Street.
1882 Godey, H. E., N. E. corner Nineteenth and Spruce Streets.
1877 Goldsborough, C. B. (N. R.)
1866 Goodell, William, 1418 Spruce Street.
1886 Goodell, W. C., 1418 Spruce Street.
1890 Grayson, C. P., 122 South Forty-second Street.
1867 Grier, Matthew J., 1531 Spruce Street.
1883 Griffith, J. P. C., 123 South Eighteenth Street.
1885 Gross, Onan B., 700 Market Street, Camden, New Jersey.
1857 Gross, Samuel D., died May 6, 1884. (O. M.)
1858 Gross, Samuel W., died April 16, 1889.
1874 Guitéras, John, 3710 Sansom Street.
1890 Gummey, Frank P., Morton and High Streets, Germantown.

1884 Haehnlen, W. F., 1421 North Broad Street.
1857 Hall, A. Douglass, resigned October 9, 1873. (O. M.)
1886 Hamaker, W. D., Meadville, Pennsylvania. (N. R.)
1891 Hamann, Carl, Cleveland, Ohio. (N. R.)
1893 Hamill, S. M., 117 South Twentieth Street.
1893 Hand, Alfred, Jr., German Hospital.
1875 Hand, Frank C., died September 1, 1881.
1885 Hare, Hobart A., 222 South Fifteenth Street.
1866 Hare, Horace Binney, died March 21, 1879.
1868 Hargadine, R. W. (N. R.)
1874 Harris, Charles M., dead.
1890 Harrison, W. H., Pennsylvania Hospital for Insane.
1857 Hartshorne, Edward, died June 29, 1885. (O. M.)
1890 Hartzell, M. B., 1411 Spruce Street.
1890 Hatch, John L. (N. R.)
1869 Hatfield, Nathan L., dead.
1879 Hazlehurst, S. F., Colorado Springs, Col. (N. R.)
1873 Hearn, Joseph, 1120 Walnut Street.
1870 Henry, Frederick P., 1635 Locust Street.
1866 Herbert, Theodore. (N. R.)
1880 Hewson, Addinell, Jr., 1508 Pine Street.
1890 Hinkle, F., Columbia, Pennsylvania.
1858 Hodge, H. Lenox, died June 16, 1881.
1871 Hoffman, Washington Atlee, died September 20, 1874.

ELECTED

1860 Hopkins, Henry St. George. (N. R.)
1877 Horwitz, Theodore, died December 12, 1877.
1870 Houston, James P. S., Savannah, Georgia. (N. R.)
1859 Hoyt, William D. (N. R.)
1891 Hughes, D. E., Philadelphia Hospital.
1882 Hughes, W. E., 3726 Baring Street.
1857 Humphries, George H., New York City. (O. M., N. R.)
1870 Hutchins, E. R. (N. R.)
1858 Hutchinson, James H., died December 27, 1889.

1890 Izard, William, Camden, New Jersey.

1891 Jamar, J. H., Elkton, Maryland. (N. R.)
1874 Jamison, Edward, St. Louis, Missouri. (N. R.)
1890 Jamison, W. B., Fortieth and Brown Streets.
1890 Jarrett, Henry, Camden, New Jersey.
1866 Jenks, William, died October 31, 1881.
1891 Jones, Charles J., 1720 North Nineteenth Street.
1885 Jurist, L. J., 839 North Broad Street.

1859 Kane, John K., Wilmington, Delaware. (N. R.)
1857 Kealer, William, resigned January 28, 1875. (O. M.)
1890 Ketcham, S. Rush, 1708 Green Street.
1889 Kimmell, Louis J. C., died 1890.
1858 King, William, dead.
1888 Kirby, Ellwood R., 334 South Fifteenth Street.
1890 Kneass, Samuel S., 261 South Fifteenth Street.
1892 Kyle, D. Braden, 1632 Chestnut Street.

1893 Lainé, D. T., Media, Pa.
1871 Landis, Henry G., dead.
1890 Laplace, Ernest, 1828 South Rittenhouse Square.
1872 La Roche, C. Percy, Nice, France. (N. R.)
1857 La Roche, Rene, died December 9, 1872. (O. M.)
1869 Leamen, Henry, 828 North Broad Street.
1893 Le Conte, R. G., 348 South Sixteenth Street.
1860 Lee, Charles C., New York City. (N. R.)
1861 Leedom, John M., dead.
1888 Leidy, Joseph, 233 South Thirteenth Street.
1887 Leopoldt, Isaac, 1518 Franklin Street.
1857 Levick, James J., resigned October 13, 1861. Dead. (O. M.)

ELECTED

1868 Lewis, Francis W., 2016 Spruce Street.
1869 Lewis, Fred. W., died 1873.
1875 Lewis, Morris J., 1316 Locust Street.
1857 Lewis, Samuel, resigned December 13, 1888. (O. M.)
1891 Lincoln, William R., 1723 North Eighteenth Street.
1893 Lindsay, John, 340 South Fifteenth Street.
1877 Little, William S., died February 17, 1887.
1886 Lloyd, James Hendrie, 3910 Walnut Street.
1886 Longaker, D., 652 North Eighth Street.
1875 Longstreth, Morris, 1416 Spruce Street.

1890 Mallet-Prevost, W., City of Mexico. (N. R.)
1874 Mann, Charles H. (N. R.)
1867 Markoe, James, died March, 1884.
1887 Martin, Edward, 415 South Fifteenth Street.
1868 Martin, George. (N. R.)
1892 Massey, Frank.
1874 Matteson, Charles C., dead.
1865 Maury, Frank F., died 1879.
1880 McClellan, G., 1352 Spruce Street.
1892 McFarland, J., 1314 Franklin Street.
1890 McKelway, George I., 255 South Seventeenth Street.
1864 Mears, J. Ewing, 1429 Walnut Street.
1873 Meigs, Arthur V., 1322 Walnut Street.
1857 Meigs, John Forsythe, died December 16, 1882. (O. M.)
1892 Miller, A. M., Bird-in-Hand, Pennsylvania.
1892 Metzler, G., 635 North Seventh Street.
1874 Miller, C. K. I., died 1878.
1882 Miller, D. J. M., 238 South Twenty-first Street.
1878 Mills, C. K., 1909 Chestnut Street.
1884 Mitchell, J. K., 211 South Seventeenth Street.
1857 Mitchell, S. Weir, resigned. (O. M.)
1893 Mitcheson, R. S. J., 1522 North Fifteenth Street.
1857 Morehouse, George R., 2033 Walnut Street. (O. M.)
1885 Morrison, William H., Holmesburg, Pennsylvania. (N. R.)
1887 Morton, S. W.
1890 Muehleck, G. A., 1320 South Fifth Street.
1871 Muhlenberg, Frank, Reading, Pennsylvania. (N. R.)
1869 Müller, August F., 4753 Green Street, Germantown.
1880 Musser, J. H., 229 South Fortieth Street.

ELECTED

1873 Musser, M. B., died March, 1888.
1869 Mustin, J. Burton, died 1871.

1869 Nancrede, C. B. (N. R.)
1891 Naylor, W. W., Lambertville, New Jersey. (N. R.)
1879 Neff, J. S., 1425 Spruce Street.
1880 Newcomer, H. W., died June 1, 1885.
1861 Norris, William F., 1530 Locust Street

1875 O'Hara, Michael, 31 South Sixteenth Street.
1891 O'Hara, Michael, Jr., 31 South Sixteenth Street.
1874 Osgood, Hamilton. (N. R.)
1885 Osler, William, Baltimore, Maryland. (N. R.)

1885 Packard, F. A., 210 South Eighteenth Street.
1893 Packard, F. R., Pennsylvania Hospital.
1857 Packard, John H., 1926 Spruce Street. (O. M.)
1875 Parks, Edward L. (N. R.)
1867 Parry, John S., died 1876.
1870 Paul, Comegys, Belvidere, New Jersey. (N. R.)
1892 Pearce, F. S., 1407 Locust Street.
1890 Pennock, W. J., 1407 North Seventeenth Street.
1891 Penrose, Charles B., 1331 Spruce Street.
1857 Penrose, R. A. F., forfeited membership by non-payment of dues, September 14, 1864. (O. M.)
1865 Pepper, George, died September 14, 1872.
1865 Pepper, William, 1811 Spruce Street.
1889 Pfouts, Gilbert B., Salt Lake City, Utah. (N. R.)
1885 Piersol, G. A., Chester Avenue and Forty-eighth Street.
1893 Posey, W. C., 1829 Chestnut Street.
1890 Potts, C. S., 1712 Wallace Street.
1886 Powell, William M., Atlantic City, New Jersey.
1889 Price, Joseph, 500 North Twentieth Street.

1885 Rahter, C. A., Harrisburg, Pennsylvania. (N. R.)
1885 Randall, B. A., 1806 Chestnut Street.
1885 Reed, Boardman, Atlantic City, New Jersey.
1859 Reed, Thomas B., died 1891.
1871 Rex, George A., 2023 Pine Street.
1864 Rhoads, Edward, died January 16, 1871.
1863 Richardson, Joseph G., died November 13, 1886.

ELECTED

1857 Richardson, T. G., New Orleans, Louisiana. (O. M., N. R.)
1884 Ridge, J. M., Camden, New Jersey.
1891 Ring, G. O., 1710 North Thirteenth Street.
1876 Risley, S. D., 1722 Walnut Street.
1868 Ritz, Charles N. (N. R.)
1880 Roberts, A. S., 1901 Walnut Street.
1876 Roberts, John B., 1627 Walnut Street.
1889 Robinson, William Duffield, 2012 Mount Vernon Street.
1876 Roland, O. (N. R.)
1874 Ronaldson, W. D. (N. R.)
1891 Rosenau, M. J., United States Marine-Hospital Service. (N. R.)
1891 Rosenthal, E., 517 Pine Street.

1867 Santee, Eugene J., 605 North Eleventh Street.
1890 Sartain, Paul J., 212 West Logan Square.
1890 Schoales, C. B., 1428 North Eleventh Street.
1890 Scott, J. A., 3808 Chestnut Street.
1874 Seiler, Carl, 1204 Walnut Street.
1869 Shapleigh, Elisha B., dead.
1885 Shober, J. B., 315 South Eighteenth Street.
1889 Shoemaker, Harvey, 107 South Twentieth Street.
1871 Simes, J. H. C., 2033 Chestnut Street.
1883 Simon, J. W. (N. R.)
1868 Sinkler, Wharton, 1606 Walnut Street.
1881 Skillern, P. G., 429 South Broad Street.
1881 Skillern, S. R., 3509 Baring Street.
1890 Sleifer, North Wales, Pennsylvania.
1890 Smith, A. Donaldson, 222 South Twenty-first Street.
1858 Smith, A. H., died December, 1885.
1888 Smith, Allen J., Galveston, Texas. (N. R.)
1863 Smith, E. A. (N. R.)
1875 Smith, Stanley. (N. R.)
1876 Smythe, F. G., died 1879.
1891 Southrn, F. L., 628 North Sixteeth Street.
1890 Stahl, B. F., 1502 Arch Street.
1889 Stengel, A., 332 South Seventeenth Street.
1889 Stevens, Arthur A., 320 South Sixteenth Street.
1886 Stewart, David D., 2620 North Fifth Street.
1883 Stiles, G. A., Conshohocken, Pennsylvania.
1857 Stillé, Alfred, 3900 Spruce Street. (O. M.)

ELECTED
1892 Stout, George C., 34 South Eighteenth Street.
1884 Stritmatter, I. P., 999 North Sixth Street.
1869 Stryker, Samuel S., 3821 Walnut Street.
1889 Sudduth, W. X., 1215 Filbert Street.

1891 Taylor, H. B., dead.
1879 Taylor, J. M., 1504 Pine Street.
1886 Taylor, L. H., Wilkesbarre, Pennsylvania. (N. R.)
1868 Thomson, William, 1426 Walnut Street.
1870 Tinkham, J. H., United States Navy. (N. R.)
1892 Toulmin, H. B., 4009 Chestnut Street.
1866 Townsend, George D. (N. R.)
1868 Townsend, R. M., dead.
1864 Turner, A. P. (N. R.)
1863 Tutt, Charles Pendleton, died May 11, 1866.
1863 Tyson, James, 1506 Spruce Street.
1890 Tyson, T. Mellor, 1506 Spruce Street.
1893 Tucker, J. A., Jefferson College Hospital.
1892 Tunis, J. P., 129 South Eighteenth Street.

1871 Valdivieso, R. (N. R.)
1891 Van Buskirk, F. W., Pottstown, Pennsylvania. (N. R.)
1887 Vansant, Eugene L., 1929 Chestnut Street.

1891 Wallace, James, 123 North Sixteenth Street.
1890 Walter, Charles, died 1891.
1890 Warder, Charles B. 1401 North Sixteenth Street.
1874 Warder, W. H., 1212 North Broad Street.
1889 Wells, George M., Wayne, Pennsylvania. (N. R.)
1875 West, Franklin, dead.
1887 Westcott, T. S., 108 North Nineteenth Street.
1885 Wetherill, R. B., Lafayette, Indiana. (N. R.)
1885 Wharton, H. R., 112 South Eighteenth Street.
1893 White, F., Elwyn, Pennsylvania.
1873 White, J. William, 1810 South Rittenhouse Square.
1893 Whiting, A. D., German Hospital.
1893 Widmann, L. A., 1324 North Fourth Street.
1868 Willard, De Forest, 1601 Walnut Street.
1889 Wilmarth, A. W., State Hospital for the Insane, Norristown.
1882 Wilson, C. M., dead.
1869 Wilson, James C., 1437 Walnut Street.

ELECTED

1869 Wilson, J. H. (N. R.)
1891 Wilson, Samuel M., 1517 Arch Street.
1891 Wilson, W. R.
1890 Wolff, L., 333 South Twelfth Street.
1890 Wood, A. C., 214 South Fifteenth Street.
1865 Woods, D. Flavel, 1501 Spruce Street.
1853 Woodward, J. J., U. S. A., died 1884. (O. M.)

1876 Ziegler, G. W. (N. R.)
1892 Ziegler, Jacob L., Mt. Joy, Pennsylvania. (N. R.)

CORRESPONDING MEMBERS.

ELECTED

1866 Bumstead, Freeman J. Died 1879.

1859 Clark, Alonzo, Professor of Theory and Practice of Medicine in the College of Physicians and Surgeons, New York.

1859 Dalton, J. C., Professor of Physiology in the College of Physicians and Surgeons, New York. Dead.

1885 Dent, Clinton T., Assistant Surgeon and Lecturer on Practical Surgery at St. George's Hospital, Surgeon to Belgrave Hospital for Children, London, England.

1860 Ellis, Calvin. Died 1883.

1888 Fedeli, Gregorio, Rome, Italy.

1858 Flint, Austin, Sr. Died 1886.

1890 Gibbes, Heneage, Professor of Pathology in University of Michigan, Ann Arbor, Michigan.

1858 Hammond, William A., Member of the National Academy of Sciences, New York.

1860 Isaacs, Charles E., Brooklyn. Died June 16, 1860.

1878 Jackson, J. B. S., Boston. Died 1879.

1886 Pye-Smith, P. H., Physician and Lecturer on Medicine at Guy's Hospital, London, England.

1860 Reeves, James E., Wheeling, West Virginia.

1861 Rokitansky, Carl. Died 1878.

1860 Watson, John, New York. Died June 3, 1869.

1859 Worthington, Wilmer, West Chester, Pennsylvania. Died 1873.

REPORT.

I. THE OSSEOUS SYSTEM.

1. *Tubercular lesions of the bones and joints.*

A discussion led, at the invitation of the Society, by Drs. JOHN ASHHURST, JR., and DE FOREST WILLARD.

DR. ASHHURST opened the discussion by saying: Chronic disease-processes of bones and joints may be divided into four groups: (1) traumatic, (2) strumous, (3) tubercular, and (4) syphilitic, leaving out of consideration malignant disease—cancer or sarcoma; certain other infectious processes, as actinomycosis; also the various forms of perforating ulcer, which all eventually involve the bones, and other conditions like mycetoma or the "fungus foot" of India, etc. Though we find cases belonging to each of the four groups separately, it is more common to find a combination, such as traumatism superadded to struma, tuberculosis, or syphilis. The tendency of modern pathology is undoubtedly to narrow the limits of struma or scrofula, and to enlarge that of the tubercular process, but it still seems desirable and necessary to retain the name struma, for there are cases which cannot be ascribed to either syphilis or tuberculosis, and which must be classified as scrofulous, or, better, as strumous. Syphilis involving the bones, whether in conjunction with traumatism or not, may be of the acquired form, but is more frequently met with in congenital cases. Finally, in tuberculosis, the disease usually pre-exists somewhere in the body, active or latent, and by traumatism is localized in the bones and joints. The traumatism in these cases plays no part except to localize, by furnishing a suitable soil for the tubercular processes.

Formerly, when this society was still young, tuberculosis was regarded as a general disease of which the lesions were but local expressions, and at this time scrofula was looked upon as an external indication, a safety-valve as it were, of the constitutional vice, to be welcomed as an element of hope rather than of danger. Soon after this period came the publi-

cation of that epoch-making book, certainly one of the most remarkable works of modern times, Virchow's *Cellular Pathology*, and following its appearance many changes in the prevailing views of pathologists. Virchow demonstrated very clearly that many processes formerly considered tubercular were not so in reality, and particularly that foci of caseation, the "crude yellow tubercles" of Rokitansky, were very often merely results of chronic inflammation. Tubercular processes were to be limited, according to Virchow, to the miliary tubercle. Villemin next showed the contagiousness of tuberculosis by injecting into animals the tubercular products, but his experiments were soon forgotten, largely because other experimenters, using various substances not derived from tubercular sources, also produced the disease, though with our present knowledge we may feel sure that their material must have been contaminated with the tubercle bacillus. Another epoch in the history of tuberculosis, almost a revolution, was brought about by the investigations of Niemeyer, who maintained absolutely that the tubercular process was a result of inflammation; and declared that in cases of pulmonary consumption the patient was first phthisical, and only subsequently became tuberculous. Then followed the experiments of Waldeyer in the artificial production of tubercular from inflammatory tissue, and the observations of Burdon Sanderson, showing the analogy between the secondary foci in tuberculosis and the metastatic deposits in pyæmia; and at the same time Sayre was proclaiming, from the clinical point of view, the traumatic origin of chronic diseases of the joints, and pointing out the undoubted fact that these cases were often amenable to local treatment, though he was wrong, according to our present views, in believing that they were exclusively the result of injury, and in not realizing that it was because the patients were tubercular that the traumatism produced the effect which it did.

At the present day all pathologists are united in ascribing to the tubercle bacillus the causation of what we recognize as tubercular processes, and we are interested only in studying how the bacillus reaches the bones and joints, and how it is brought into operation. At one time it was thought that all chronic diseases of the articulations, and especially of the hip, were tubercular, and no treatment beyond palliation was attempted. After the revulsion caused by Virchow's teachings a directly opposite view obtained, and but few cases were regarded as tubercular, the great mass being looked upon as purely traumatic. Some observers still held to the existence of a strumous diathesis, and we cannot, it seems to me, help believing in the presence of some predisposing cause in such a case, for instance, as the following: A child, in

playing on the ice, fell and sustained a bruise on the elbow; instead of the patient convalescing promptly, arthritis ensued, pyarthrosis of the elbow-joint followed, then acute necrosis and osteomyelitis of the humerus, and finally pyarthrosis of the shoulder. After amputation at the latter articulation, recovery was ultimately obtained. Such cases point indubitably to a diathetic taint, and in many instances this may properly be termed struma; since we cannot say that all of these cases are tubercular. Struma was considered by the elder Gross as but the remnant of syphilis in the individual's ancestors, perhaps several generations back, but to-day we are inclined to look on struma as more closely related to tuberculosis, though it is probable, as Gross maintained, that some of these cases are the outcome of syphilis in grandparents or ancestors even more remote.

Traumatism, though rarely the sole cause of chronic bone or joint disease, is very frequently the agent by which syphilis or tuberculosis becomes localized in the bones or joints. It acts by so injuring the parts as to make them of low vitality, furnishing a favorable soil for the growth and development of the syphilitic or tubercular poison.

In the case of tuberculosis, the presence of the bacillus is a necessary forerunner of the disease. Tubercle bacilli may enter the body through the air, penetrating the lung substance, and finally lodging in the bronchial lymph-glands, where they may lie latent; or they may be introduced into some wound or abrasion of the exterior, to be carried by wandering cells to the neighboring lymph glands; or finally the micro-organism may gain entrance into the blood, either, as Weigert has shown, by formation of a tubercular focus in the wall of a vein, or, according to an observation of Ponfick, by implication of the thoracic duct, or, perhaps, even by involvement of an artery. It is probable, indeed, that many individuals who are in good health and, to all appearances, free from tubercular infection, carry within them, locked up in some lymph gland or out-of-the-way part of the body, a nest of tubercle bacilli, which may remain thus latent for a long time, until by a neglected cold, an uncured bronchitis, or a pneumonia, or by some injury to a bone or joint, a suitable soil is prepared for their growth and development. If the injury be slight, if the resistance of the tissues be but little impaired, or if the bacilli be but few in number, the powers of nature may succeed in throwing off the poison and preventing general infection, but if the lesion is more extensive, or the bacilli in great numbers, all of the conditions are supplied for the rapid development of the tubercular process.

Symptoms. In general terms we may say that tuberculosis is probably present in those cases of bone or joint disease in which there is rapid casea-

tion and rapid suppuration. As a result of this, sinuses are formed, and external discharge ensues; but it must be remembered that in some cases extensive destruction of parts may take place, and no sinus, or at most an apparently trifling one, be present to indicate the extensive changes below. Caries is another indication of tuberculosis, but it must not be supposed that caries and tuberculosis are necessarily connected. Caries is to bone what ulceration is to the soft parts, and nothing more. Tuberculosis tends rather to produce caries than necrosis. The tubercular process about joints usually begins in the articular ends of the bones, and not as do the pyæmic processes, such as gonorrhœal rheumatism (for this appears to be a mild pyæmia), in the synovial membranes.

In a few cases the disease may begin in the soft tissues, as in some cases of hip-joint disease, in which the process probably begins in the *ligamentum teres;* but usually tubercular disease takes the form of osteo-arthritis. When the bone immediately beneath the articular cartilage is eroded, pain becomes a prominent symptom, and as the disease advances tubercular lesions will commonly be found in other parts, as in the lymphatic glands or in the internal viscera, notably the lungs.

Differential diagnosis. 1. In what may be called *traumatic-strumous* cases of joint disease, that is such as are due to traumatism in a scrofulous person, but in which the traumatism is of greater importance than the strumous condition, fibrous ankylosis appears early, and the joint may be entirely obliterated without the appearance of suppuration at any stage.

2. In the *strumous-traumatic* cases, on the other hand, those in which the strumous element preponderates, we find the gelatiniform changes characteristic of the so-called scrofulous synovitis, which I have preferred to call "gelatinous arthritis." These cases are of an indolent character, the disease causing little pain, and not preventing motion of the joint until at a comparatively late period. Microscopically, the gelatinous tissue does not, according to Barwell, differ from ordinary granulation tissue, except that it does not, as in other forms, go on to complete organization; but Godlee's and Friedländer's observations seem to show that, in some cases at least, the gelatinous change is actually one of tuberculosis.

3. Syphilitic affections of the bones are for the most part characterized by great weight of the limb, from actual hypertrophy of the bone, which is the seat of a condensing ostitis. This process is not, however, to be regarded as distinctly syphilitic, though it frequently occurs in syphilitic cases. Sometimes osteophytes are developed. If necrosis occurs it is very chronic, and the sequestra manifest little tendency to

be cast off. Osteocopic pains are particularly marked, but are due merely to the condition of condensing ostitis, and are, therefore, not necessarily an evidence of syphilis. When the joints are affected, they become greatly swollen, sinuses are formed, and on incision much gummy material is found outside of the joint, with sometimes surprisingly little change inside of the articulation. Besides these indications, manifestations of syphilis are seen elsewhere, in the notched teeth, keratitis, or other lesions characteristic of the disease.

4. *Tubercular* cases are manifested by great pain and tenderness, with partial, sometimes bony, ankylosis in one part of the joint, and extensive tubercular changes in other parts. In the "traumatic strumous" cases the ankylosis is usually fibrous. There are also in tuberculosis rapid caseation and suppuration, with formation of sinuses, and, on incision, disclosure of extensive changes within the joint, often more advanced than would be expected from external examination. Cachexia develops in the course of time, as also lymphatic involvement, and finally perhaps pulmonary tuberculosis.

Prognosis. The course of tuberculosis in bones and joints, after the process has actually begun is unfavorable, but if the diseased area is removed early life may be saved, for bone tuberculosis is primarily local, subsequently spreading like the secondary foci in pyæmia, as Burdon-Sanderson has shown. The local lesions may be removable or not, but in all cases constitutional treatment is of equal importance with local. Either alone will not suffice. Operation is always called for in suitable cases, and if done early there is fair hope of recovery. But strongly as I advocate a resort to operation in destructive affections of the bones and joints, I believe that in no cases are Abernethy's teachings as to the "constitutional treatment of local diseases" more important than in these.

Therapeutics are beyond the domain of this Society, but I may say that in my judgment, together with constitutional treatment, intra-articular and interstitial injections of iodoform and glycerin, the cautious use of the Liqueur de Villate, simple erasion with the sharp spoon, Sédillot's operation of *évidement*, arthrectomy, excision, and even amputation, may each be properly resorted to in suitable cases of tuberculosis of the bones and joints.

Dr. De Forest Willard: The tubercular process in bones and joints, viewed either pathologically or clinically, is an inflammatory condition of low grade tending to degeneration rather than to resolution.

The discovery and recognition of the tubercle bacillus has done much to elucidate the study of this disease. Heredity and traumatism play essential parts in the causation of most cases, hence all individuals are

liable to attacks. Traumatism acts as a cause by awakening a tubercular lesion and localizing it when the bacilli are already present in the system. Bacilli are probably far commoner in the blood than we have yet any means of determining, and traumatism, by causing congestions, endarteritis, and various other processes, prepares for the bacillus a suitable soil for growth and multiplication.

The disease—tuberculosis—is unquestionably a local one, arising easily in those of tuberculous diathesis; with great difficulty in those who are not so constituted, and who are therefore able to ward off the incursions of the bacillus. Practically, all persons are exposed to traumatisms, and hence to the dangers of invasion by this process, and escape depends only upon the resistive power of the individual. I am a strong believer in the effect of heredity. I believe that an individual receiving such a taint from his ancestry, no matter how remote, has less capability of resisting invasion from these bacilli than has a person with no such transmitted tendency. No one can deny that this condition arises *de novo;* nor, on the other hand, can any one positively deny the non-existence of hereditary taint. It is of the utmost importance to emphasize to the profession the fact that a local tubercular process can originate in an individual, since many physicians have thought that a given case of joint disease cannot be tuberculous, simply basing their decision on the fact that they have not discovered evidence of tubercle in the father or mother.

Inquiries upon the question of heredity are nearly always met by absolute denial on the part of the patient, no matter how apparent the truth may be. I have frequently failed absolutely to elicit a trace of tuberculous antecedents, when a year later, or a closer acquaintance and observation, demonstrated most completely the existence of heredity. Whether such a condition exists in the individual or not is of little consequence when we see positive evidence of tuberculous disease before us. The only advantage derived from a knowledge of such a taint is the increased vigilance and care which must be exercised by the surgeon both in the prevention of traumatism and in the length of the continuance of treatment.

Formerly a few contended that traumatism was the sole cause of this process, and that hereditary tendency had nothing to do with its production. At present few care to stand upon such ground, since, although traumatism is almost universally acknowledged to be the exciting cause, yet the underlying element, the non-resistive force, is also universally recognized. An individual without hereditary taint or tendency to degenerative inflammation receives a blow; inflammation

follows, goes on to resolution, and the parts resume their natural functions; the same traumatisms in persons of non-resistive power ends in destructive ostitis.

Again, a bone or joint perfectly competent to ward off the invasion of an enemy under ordinary conditions loses its resistive power under the stagnation and lowering influence of inflammatory action following an injury, and degeneration is the result.

This inherent, systemic, inhibitive power is an element most constantly powerful and effective in the normal tissue. Multiple foci of tuberculous invasion may be limited, encapsulated, warded off, or absorbed. In numerous instances I have seen three, four, or even six separate joint involvements, yet victory has been gained over the disease at last. In these multiple invasions everything depends upon the resistive power of the individual. If weakened by disease, by hereditary influences, by traumatism, by inflammatory conditions, by any depressive force, the bacilli may gain the ascendency.

The tubercular process pathologically considered presents a slow, chronic inflammatory process, characterized by the presence of a special microbe, with a tendency to degeneration, caseation, liquefaction, or suppuration. The decade which has passed since Koch demonstrated the special microbe concerned in the production of tuberculosis has been one of steady, though slow advancement in accurate pathological knowledge of this disease. Although the old, complex clinical term "scrofulous" was then, and still continues to be, most practically indicative of this process of degenerative action, yet if we consider scrofulosis and tuberculosis one and the same pathological condition, clinically we shall be better able to combat the ravages of the process. Koch demonstrated not only the presence of this specific bacillus, but also by culture tests and by inoculation proved the absolute truth of his former theories.

The work of the older pathologists in this direction should not be overlooked. The inoculation experiments of Kortum, carried on a century since, yielded tardy fruit, yet were important. They were elaborated and developed in their successive stages by Villemin, Cornil, Klebs, Max Schüler, Sanderson, and others. Although these men did not actually demonstrate the existence of a bacillus, or inaugurate the crucial test from pure cultures, yet their work was valuable. They proved that true tuberculous products, especially the gray granulations, are easily inoculable either by injections hypodermatically or by inhalation. Slowly caseating masses are the most contagious, but if rapidly degenerating they are the most virulent.

Pseudo-tuberculosis is not uncommonly seen, and it is this process that has been produced by Weichselbaum, Martin, and others, by inhalation of pepper, cheese, etc. (The only wonder is that Limburger did not do worse!)

The explanation of the tuberculous process, of the transition of an inflammatory process into a tubercular one is often found in the congestive hyperæmia from injury. An inflammatory endarteritis of the Haversian canal easily blockades the bacilli, causing stagnation, degeneration, absorption of the trabeculæ, deposit of tuberculous material, softening of the centres of the mass, followed by caseation or by suppuration. A tendency to tuberculous degeneration of the bone early invites bacillary infection, and if the parts are already enfeebled by hereditary tendencies the microbes gain the ascendency. Rarefying ostitis having been established, absorption of bone follows, the spaces are enlarged, fatty degeneration follows, and the contents are transformed into granulation tissue.

A mass of gray tubercle may become yellowish in the centre, break down, and either work its way into a joint and degenerate into pus, or it may caseate. Though caseated, it still contains bacilli, which, though dormant, may be aroused into activity by traumatism or by depressing disease.

I have in a number of instances struck into these old caseated deposits while performing late osteotomies, or myotomies, with the result of awakening slight and short-lived suppuration only.

It should be remembered that the natural tendency of tubercular degeneration is not toward the formation of pus. The latter tendency is usually the result of a superadded inflammatory process. Hence the value of those methods of treatment which aim to prevent traumatism, friction, and all causes of inflammation. The tubercle bacillus and the pus microbe are not the same. In the one case there is coagulation, necrosis, caseation, and liquefaction; on the other hand, suppuration.

Another point to be borne in mind is the fact that an abscess wall, the old "pyogenic membrane," is a bulwark of protection formed by embryonal cells to shut out the offending agent from the general system.

The tubercle bacillus seems largely to diminish in the presence of suppuration, and sometimes cannot be discovered.

The first step in degeneration is always decreased vascularity. Fungous granulations often seen in an old tumor albus at the knee, or at the wrist or ankle, are excellent illustrations of the growth of this tubercular tissue. The presence of the yellowish-white tubercules in the

deeper granulation tissue is ordinarily evident. A joint usually becomes infected by perforation into its substance or by extension from the original focus. Occasionally the articulation seems to be affected before actual perforation occurs by an extension of the inflammatory process. The synovial membrane inflames; the cartilages are seldom primarily affected.

In bones the process frequently begins in the epiphysis because this is the growing, the active part of the bone, and hyperæmias are most frequent in this situation; but, later, extension in different directions occurs, and there may be perforation into a joint, secondary infection of the synovial membranes, and joint tuberculosis. At the hip the epiphyseal line is within the capsule, and the disease easily reaches this articulation. The influence of tuberculosis in producing bone and joint disease has been amply demonstrated by the identity of the process as regards the same condition in other parts of the body—by inoculation and by culture experiments.

Although the synovial membrane and the epiphysis are entirely separate structures, and although the tubercular process may primarily affect either, and although the initial focus of a large majority of cases lies in the latter region, yet the ultimate results are practically the same. In either situation the course of the process will be: first, local congestion, stagnation; then degeneration and the formatiou of little yellow spots (tubercles); these spots in turn softening in their centres by a suppurative process, particularly if the inflammatory process is superadded, or else undergoing a process of resolution. The march toward resolution consists of caseation with or without liquefaction, and indicates the victory of the phagocytic processes over the bacilli.

In the present stage of pathological research, and until the laboratory shall disclose some better explanation, we must admit that a certain bacillus is the cause of the tubercular process. Practically we know that some destructive element is at work, whether it be a bacillus or other agent, and we recognize that this element is powerful to destroy unless overpowered by the vitality inherent in the body alone, aided by the physician's drugs or the surgeon's knife.

Practically the interest of the surgeon concentrates upon tuberculosis of the joints, bones, and glands. In joint tuberculosis the non-existence of bacilli does not necessarily indicate the absence of this process, since the inflammatory element may be in the ascendency, or caseation may be slowly progressing, although clinical features are sufficiently distinctive to assure a diagnosis. A careful microscopist, however, will usually

discover some evidences of the microbe either in giant cells (macrocytes), or between the epithelioid cells (platycytes).

It is very fortunate that these tubercular cells are not migratory of themselves, else rapid systemic invasion would be the rule. They may, however, be carried forward by migratory inflammatory cells, and thus disseminate the disease. In cheesy deposits it is rare to find bacilli. In these deposits, however, we do find spores, which may be disseminated and then become virulent.

Multiple foci of tubercular disease are of great interest. Thus the hip and spine, or both, may be affected, and yet no involvement of the lungs may occur and the patient may be in fair general health. The depression of the patient in later stages is not infrequently produced by secondary infection, by the micro-organisms of suppuration entering through sinuses and setting up extensive suppuration. So, too, caseation is a secondary process, and follows after the true tubercular processes.

It is difficult to determine in cases where the foci of this disease are multiple whether the systemic inflammation follows in consequence of expansion of the process from the original seat, or whether the resistive power of the individual is steadily undermined, giving ascendency to the disease. I am treating at the present time six cases of tubercular disease in both hips; a large number of others in which a hip and one or more vertebræ have been the seat of the disease; one in which the shoulder, the humerus, and both hips are implicated; another in which both hips, one knee, and both wrists are diseased; and another where the wrists, elbow, spinal vertebræ, and tarsus are the seat of the foci; and still another in which both knees, both shoulders and elbows are tuberculous. In these cases a secondary focus frequently develops when the patient is lying absolutely in bed, and traumatism can, therefore, be excluded as a causal element.

Cutaneous Inoculation. While it is contended that the epithelium is perfectly capable of resisting this virus, and though this is undoubtedly true, yet an abraded surface is non-resistive. I well remember the case of an elderly lady who was nursing her grandchild, suffering from tuberculosis, where the former became inoculated through a sore upon her finger. This went on to ulceration, broke down, and continued stubbornly to resist all forms of treatment. Finally the glands in the axilla became involved, then those above the clavicle and in the neck, until at last she died from exhaustion. The tubercular nodules in her neck were very marked.

Although this is a pathological discussion, I cannot refrain from

drawing a few practical and clinical conclusions concerning this process in bones and in joints.

First. Traumatism in the region of the joints in children is especially dangerous and is frequently followed by evil results. Cases, therefore, of so-called sprain, contusion, pain simulating rheumatism, and other like affections, especially about the hip and knee, should always be regarded with suspicion, and be treated with a full knowledge of the possibility of a subsequent degenerative process.

Second. Cases with inherited tubercular tendencies should be especially watched after any inflammatory condition in the region of the articulations; the diagnosis and prognosis of these cases should be guarded, and the supervising care should be long continued and critical.

Third. As the inflammatory action is not upon the surface, but is concealed from sight, double caution should be exercised in the treatment of the few signs of chronic inflammation which are present.

Fourth. All cases of doubt had best be treated upon the hypothesis that they are tubercular, since no harm can thereby be produced, and many a joint will be saved by such a course.

Fifth. Inflammatory conditions, acute or chronic, yield better to rest than to all other forms of treatment combined. If rest is instituted early the hyperæmia is relieved, inflammation is aborted, and the case goes on to resolution instead of advancing to tuberculous degeneration. Early treatment, therefore, during the first ten days of inflammatory conditions is of the utmost consequence. The earliest signs of tubercular inflammation are discomfort in the joint, and restlessness of the limb at night. These antedate rigidity, as rigidity so frequently antedates pain.

We may with justice deduce from our studies of tuberculosis of the bones and joints that it is a local process, and that early removal of the diseased focus may prevent general infection. Sometimes such removal is easy; in other cases impossible, as when dissemination has already occurred or there are multiple foci; again, by the very operation of removal new tissues may be opened up for further infection. The last is a serious danger. Another deduction is that a person of tubercular diathesis should at all times guard against injuries of the bones, and that after any injury the most rigid and prolonged care must be observed lest the tubercular process be initiated. *February* 25, 1892.

2. *Tubercular arthritis of left hip, with death resulting from general infection through the blood and lymphatic system.*

Exhibited by Dr. J. C. Bloodgood.

S. B., a colored girl, aged 11 years, was admitted to the Children's Hospital in February, 1891, and died six months later. No family history; parents and one sister living. Her health began to fail when she was 9 years old, and hip symptoms three months before admission. She was in the second stage of coxalgia; no suppuration, and very much emaciated. During the six months her temperature was always hectic, 97° to 104°; pulse and respiration rapid. No coughing or expectoration. No diarrhœa until the last few weeks; then slight looseness and tympany, but no great abdominal tenderness. No vomiting, and pain only in hip and over sacrum.

Post-mortem. Very much emaciated. Capsular ligament of left hip distended with curdy pus. The ligamentum teres was gone and the periosteum covering the head of the femur thickened, and the bone was enlarged, soft, and cancellous. Trochanter, neck, and shaft of femur seemed normal. A large pocket of pus below and behind capsule, probably a bursa. Peri-articular tissue (fibrous) thickened and increased. The lymphatic glands in Scarpa's triangle were enlarged and presented cheesy necrosis. The lymphatic vessels and those glands following the left iliac vessels and the abdominal aorta into the posterior mediastinum are all involved, and the glands of the mediastinum enlarged and in cheesy necrosis. The same condition was found in the lymphatics going to the liver and to the spleen; the spleen was dotted with white nodules of various sizes up to a pea, and also two peripheral hemorrhagic infarcts were present. The mesenteric glands were enlarged and cheesy, the great omentum dotted with white pin-head nodules; the liver filled with white pin-head nodules through its substance, but chiefly under the capsule; the kidneys, bladder, and pancreas seemed normal. The peritoneal cavity contained ten ounces of milky fluid.

The pleura of the left lung was so adherent and thickened that it was removed with the lung. The entire left lung, except the apex, was solid, with large, cheesy masses. A distinct barrier of fibrous induration with reddish edges separated this portion from the apex, which contained a few nodules and one small cavity. The right pleura was not adherent, but the lung contained a few white nodules of small size. The heart, larynx, and brain were normal. No microscopic study was made.

Conclusion. It was impossible in this case to tell the initial seat of infection. Had it been the hip and had the case been seen early, an operation might have prevented further infection. Such general infection associated with hip disease is not common; this is the only one on record for many years at the Children's Hospital. *March* 10, 1892.

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3. *Fracture of the spine and ribs.*

Exhibited by Dr. John B. Deaver.

The specimen exhibited showed a complete fracture of the spine in the dorsal region, with fracture of several ribs on each side. The patient, a woman, had fallen from the third story of her house and struck upon the back. Shock was profound, and she died the next day. At the autopsy the fractures indicated were discovered, and it was found that the spinal cord was severed transversely. This was remarkable from the fact that there had been no apparent deformity. The transverse division could not have been complete, for slight movement of the feet and legs had been possible before death. The specimen illustrates the uselessness and danger of operation in many of these cases, for there is no manner of determining definitely the condition of the cord, and even though there be little or no deformity, complete transverse destruction may be observed.

Dr. Wharton agreed with the speaker in regarding the specimen as a warning against operation in many cases of spinal fractures.

Dr. Meigs referred to a case of his in which there was complete paraplegia and no injury of the spine was discoverable. Operation was therefore not performed. At the autopsy a complete traumatic division of the cord in the cervical region was detected. *December* 8, 1892.

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4. *Osteo-sarcoma of the tibia.*

Exhibited by Dr. H. B. Allyn.

The patient was a woman aged 79 years. The previous history had no bearing on the case. Three months before she came under observation she had fallen and broken her leg at the lower third of the tibia, but the tumor sprang from the upper third, and the injury is therefore

of doubtful significance. At first there was irregular pain, increasing with attempts at motion, and sometimes subsiding wholly. There was no heat of the part and no swelling. Several weeks later the part assumed inflammatory appearances, and the case was regarded as one of ostitis. Gradually the head of the tibia grew larger and several points became prominent and tender. At times there was considerable œdema and redness. Death occurred nine months after the first pain. The tumor is seen to spring from the tibia and is a typical osteo-sarcoma.

January 12, 1893.

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5. *Specimen of elbow, with fracture of the external condyle of the humerus; chipping off of the coronoid process of the ulna and anterior lip of the head of the radius.*

Exhibited by Dr. G. G. Davis.

The arm exhibited was removed by Dr. Patek at the German Hospital, for compound comminuted fracture of the shaft of the humerus. The patient was a man aged 25 years, who had been run over by a street car. On examining the elbow-joint a fracture is seen extending from above the external condyle diagonally through the capitellum and just involving the external edge of the trochlea. These fractures of the elbow-joint are interesting on account of the difficulty which exists in successfully treating them. A displacement upward of either the internal or external condyle will, if the fragment unites in that position, cause a deviation in the normal axis of the limb. When the normal elbow is extended, the hand being in supination, the longitudinal axis of the forearm is not coincident with that of the arm, but forms an obtuse angle with its apex inward. An increase in this angle, or the occurrence of an angular displacement inward constitutes what is known as "gunstock" deformity. It is more common after injuries of the internal than the external condyle, and I would like to call attention to the anatomical relations of the parts as accounting for this fact. The radius is comparatively loosely attached to the humerus; in the movements of pronation and supination it alone moves while the ulna is stationary. The ulna, on the contrary, articulates firmly with the humerus by a purely hinge-joint that is prevented from being displaced by its being concave from side to side. As the forearm and hand are supported almost solely by the ulna any injury to the internal aspect of the joint with which it articulates (the trochlea) is far more liable to be followed by displacement, the looser connections

of the head of the radius to the humerus (the capitellum) not sufficing to retain the normal axial direction of the limb.

Associated with the fracture of the external condyle already described, was a chipping off of the anterior lip of the head of the radius and also of a small portion of the apex and outer surface of the coronoid process of the ulna. It may hardly seem right to designate such apparently slight injuries as fractures, but they constitute the typical lesions ordinarily produced by backward dislocation of the bones of the forearm or the arm only; they are oftentimes greater or more marked in extent. On the anterior portion of the head of the radius is seen a rough surface from which a scale of bone about three-eighths of an inch square has been knocked off. On the coronoid process is a similar rough surface, triangular in shape, about a quarter of an inch in size, and above it are two small loose pieces of bone. It will be seen that these detached fragments are still firmly adherent to the anterior portion of the capsular ligament, and it shows that in fractures of this process the persistence of this connection not only aids in preventing marked displacement, but also insures a sufficient supply of nutriment to the fragment, and thus enables it again to unite with the shaft of the bone. These lesions of the head of the radius and coronoid process are not by any means common, and, though not as marked as they might be, are still typical of their kind. *May* 11, 1893.

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6. *Fracture of the posterior lip of the carpal extremity of the radius (Barton's fracture), produced post-mortem.*

Exhibited by Dr. G. G. Davis.

On the carpal extremity of the radius is a very interesting lesion which I produced post-mortem. It is a fracture involving only the posterior lip of the carpal extremity of the radius, a so-called Barton's fracture. It was produced by resting the dorsum of the hand on a flat surface and violently flexing the wrist by pressure on the bones of the forearm. It occurred while endeavoring to produce a Colles fracture by palmar instead of dorsal flexion. In cases of Colles fracture I have so often had the history given me of the injury having been produced by palmar instead of dorsal flexion, that I was desirous of seeing if I could so produce it; the fracture just described was the result. It will be seen that there is a thin shell torn off of the posterior lip of the radius; it is about five-eighths of an inch long, extending five-sixteenths

of an inch down on the articulating surface and extends about a quarter of an inch up the radius. The injury is certainly rare in surgical practice, and has been principally observed in connection with backward dislocation of the carpus. Stimson thinks it should be regarded as a complication of dislocation instead of a true fracture; but this specimen proves that dislocation is not necessary for its production. Recently I have had two fractures of the lower end of the radius, one in a boy aged 10 years, and the other in a girl aged 13. In both cases the patients were positive that the injuries occurred while the hand was in the position of palmar flexion. In both there was abnormal mobility, crepitus, and pain, and in neither was the styloid process included in the fractured part, so that the fragment must either have included the whole thickness of the bone, yet not its entire width, or else must have entered the articular surface. Both my resident, Dr. Doane, and myself regarded the case of the boy as not embracing the entire thickness of the bone and as being a true Barton's fracture. There was no pain on the palmar surface of the wrist nor any other indication that the injury had penetrated that far. In the second case we were unable to satisfy ourselves positively whether the whole thickness of the bone was broken or not, but we were positive that its whole width was not, and that the styloid process was uninjured. *May* 11, 1893.

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7. *Specimen of ungual exostosis.*

Exhibited by Dr. G. G. Davis.

This specimen is the end of a big toe removed for an exostosis of the ungual phalanx. On examining it there is seen projecting from beneath the nail a hard, rounded tumor about three-eighths of an inch in diameter. A longitudinal section shows that it is an exostosis proceeding from the upper surface of the ungual phalanx; it projects forward beneath the matrix of the nail, and has its extremity covered by fibrous tissue. Posteriorly it merges with the periosteum and bone at about the root of the nail. It came from a young woman aged 23 years, who had had it about four years, and she attributes its presence to having worn shoes which were too short. She said that the pain at the posterior portion of the nail was quite marked, and it is probable that it was started from the irritation caused by the pressure of the nail backward. This affection seems to be most common in young women, as it is the second case in which I have operated for it, and it is strongly suggestive that the explanation as to its origin above given is the correct one.

May 11, 1893.

8. *Ostitis of the head of the femur.*

Exhibited by Dr. H. R. Wharton.

Dr. Wharton exhibited the head and neck of the femur removed from a child aged 6 years. The process began as a primary ostitis of the head of the bone, and there was a distinct sequestrum, which was shown in the relation held before removal.

A study of a series of such cases demonstrates that a majority begin in the bone, and not, as has been held, in the synovial membranes. The cartilages in this case were little affected. *May* 25, 1893.

9. *Fracture of the pelvis and laceration of the bladder.*

Exhibited by Dr. G. G. Davis.

A. A., female, aged 32, was injured by being struck and pushed ahead by the wheel of a cable car. It passed directly between the thighs striking the perineum. The internal genitals were very badly lacerated, which laceration extended into the vagina. There was evident fracture of the ascending and horizontal rami of the left pubic bone, and she had hæmaturia and was in marked shock. Death ensued in twelve hours.

Autopsy disclosed fracture of the left horizontal and ascending rami through the thyroid foramen and the left sacro-iliac synchondrosis. There was also a fracture of the sacrum extending from the upper left corner down and outward, and also a fracture of the coccyx. A small laceration was found in the walls of the bladder due to perforation by the fragments of the horizontal ramus. *June* 22, 1893.

II. THE DIGESTIVE SYSTEM.

1. *Specimens from a case of typhoid fever with purpura hemorrhagica.*

Exhibited by Dr. Allen J. Smith and Dr. F. S. Pearce.

The subject from whom the specimens here presented were obtained, J. C., a white laborer, aged 21 years, single, was admitted to the Presbyterian Hospital on August 11, 1891, with the history that eight days previously he had experienced a chill, followed by two or three attacks of epistaxis. He had flashes of heat, and following them periods of profuse perspiration. His bowels from the time of the initial chill were inactive, and the patient very much constipated. Upon admission his temperature was 103.2° F.; pulse, 100; respiration, 28. The patient was from the first disposed to present nervous symptoms. Throughout the course of the case the urinary examination was negative. By 9 p.m. of the day of admission the temperature had reached 104° F., but fell to 101° F. by the following morning, with a pulse of 80 per minute and respirations 24 per minute. The characteristic abdominal spots of typhoid fever were well developed at the time of admission. By sponging the temperature was kept, as a rule, below 104° F.; the highest temperature was reached on the second day after that of admission at 5 p.m., 104.2° F. The bowels were moved once each on September 12th and 13th, the passages being of a semi-liquid consistence and light yellow color. He vomited milk taken on both these days. On September 14th there were two stools; otherwise the patient appeared to be doing very well. On the following day the pulse began to assume an irregular character, varying from 62 to 80 per minute; and again there were two stools. He rested quietly through the night of the 16th, and had a semi-liquid, yellow passage from the bowels on the morning of the 17th. On September 16th there was some epistaxis, not sufficient to annoy the patient, until in the evening, when Dr. Pearce, the resident physician in charge of the case, plugged the posterior nares, with happy result. However, on September 17th, the bleeding again started in, numerous expedients being resorted to before the hemorrhage could be stopped; again at 5 p.m., the epistaxis became alarming and he vomited a large amount of blood. Hæmatemesis con-

tinued profusely at intervals until death. At 9 P. M. on September 17th, a cold perspiration broke out over the entire body; the temperature at midnight was 96.1° F.; pulse, 112; respiration, 24. Ergot and morphia were administered, hot bags were applied to the feet, and other measures were taken to bring about reaction. At 1 A.M. of the 18th the temperature was normal; at 2 A.M. it was 100.6° F., the pulse very weak and ranging from 136 to 160. The patient experienced sensations of great thirst, and was very nervous and restless, the cold perspiration rolling from him for six hours previous to dissolution. He died at 6 A.M., seven days after his admission to the hospital and on the sixteenth day from the initial chill.

It was noted after death that over the chest there were here and there purpuric spots, although none had been noticed previously.

At *autopsy* little or no emaciation was noted. Over the skin of the abdomen there was a fine purpuric eruption, the spots brightly livid and most numerous over the left side of the abdomen. Here and there over the chest and limbs there were also a few spots. The largest of these, one over the left clavicle, was about one-fourth of an inch in diameter. On section, the pleural cavities were free of fluids and the membranes of adhesions. On the anterior surface of the left lung there were purpuric patches larger and darker than those of the skin, and on cutting into the pulmonary structures similar areas of hemorrhage were found. The right lung was entirely free of these hemorrhagic patches. Both lungs were the seat of hypostasis at the bases and about the roots. The heart muscle was dark in color and very flabby; there were no abnormalities of the endocardium of the cavities or valves. In the visceral pericardium there was noted a small patch of purpura on the anterior aspect of the heart. There were numerous patches of ecchymosis in the peritoneum, both that covering the intestines and that lining the abdominal cavity. There was no evidence, however, of any inflammatory change in the membrane. The right lobe of the liver was covered with a dense purpuric eruption of the general appearance of that seen in the skin, but darker in color; however, there were no areas in the hepatic tissue as those seen in the pulmonary structure. The substance of the liver was soft and friable. The spleen was decidedly enlarged, measuring about six inches in length, four in width, and three in thickness. The substance was dark and very soft. The left kidney was small and dark in color; the capsule was removed easily. The cortex was of the usual thickness. Beneath the mucous membrane of the pelvis of the organ there was collected a mass of dark, clotted blood, from one-half to three-fourths of a fluidrachm in amount.

The right kidney was normal. The mucous membrane of the stomach was the seat of numerous and closely placed purpuric patches; and the organ contained about half a pint of grumous fluid. The mucous surface of the intestines was less densely mottled, but contained, besides the small spots, extensive areas of hemorrhagic suffusion. Near the ileo-cæcal valve and a short distance above and below were to be seen small, rounded ulcers, the largest not over one-half or three-fourths of an inch in diameter. In one or two places the ulcers were elongated, their long axis transverse to that of the gut; and in all cases the margin of the ulcer was thick, masses of whitish slough marking the central portion. The mesenteric glands were all enlarged and of a dark-red color. There was no evidence of tubercular deposit in any of the organs. The blood was dark and fluid, the clots in the heart being insignificant.

The specimens shown in this case were the left kidney and the intestine, exhibiting the appearances as described, but in a somewhat altered condition owing to the bleaching action of the alcohol preservative upon the color of the tissues. *September* 10, 1891.

2. *Specimens from a case of typhoid fever, with intestinal perforation and peritonitis.*

Exhibited by Dr. M. Howard Fussell.

The patient from whom these specimens were removed, James B., was 25 years of age, married, and the father of one child. He was admitted to the exhibitor's ward in St. Timothy's Hospital, September 7, 1891. The following history was obtained: He was English and had been in this country one year. Family history was negative. He had enjoyed good health as a rule, but had an attack of abdominal pain six months ago, which confined him to bed for several days. His condition at that time was diagnosed as "bilious dysentery" by his physician.

Three weeks before admission to the hospital he began to complain of malaise, pain in the limbs and general weakness. He had no diarrhœa at any time, and no delirium. He had been in bed two weeks before admission and was being treated for typhoid fever. On the date of admission (September 7, 1891) at 5 P.M., his temperature was 105° F. He was sponged and given half an ounce of whiskey and a dose of salol every three hours. By the next morning, when the exhibitor first saw the patient, his temperature was 103.2° F., with a weak pulse of

110 beats per minute. His face was not flushed but somewhat glazed; tongue dry; slight meteorism; some tenderness in right iliac fossa. Splenic dulness extended over a decidedly increased area. The heart's action was rather weak, with a faint sound suggesting the presence of a murmur.

The patient steadily improved, his pulse becoming better, and his temperature gradually falling until September 11, 1891, when it was registered as 101.8° F., in the evening. On this day he had three fluid stools. On the following morning he complained of slight pain in the abdomen, and about 6 A.M. became suddenly collapsed. At 10 A.M. when the exhibitor saw him, he was almost pulseless, his skin covered with a cold sweat, his abdomen tense, full, and not very painful, and his temperature 101.5° F. At noon the latter had gone up to 102° F. The patient was conscious and did not complain of much pain. He never reacted, however, and died at 2 P. M., eight hours after the appearance of collapse.

Post-mortem examination was held forty-eight hours after death. On opening the abdomen the coils of small intestine were seen to be brightly injected and red, the color being due to the injection of the acutely inflamed peritoneum and not to the filling of the vascular walls of the intestine. The surface in many places was covered with soft lymph, and there was some lymph also upon the parietal peritoneum. In the abdominal cavity there was considerable bloody, fecal fluid, filling the pelvis and measuring from ten to twelve fluidounces. The omentum was adherent in the ileo-cæcal region by old and firm attachments; the appendix and cæcum were fixed in a mass of old adhesions. The appendix was small and apparently healthy in spite of its adhesion on every side. The mesenteric glands were enlarged and softened. The intestines contained considerable soft, semi-fluid fecal matter, but no blood. In the ileum about a foot from the ileo-cæcal valve there was a perforation extending through all the coats of the intestine about the size of a silver dime. The Peyer's patch, in the centre of which this perforation was, measured about an inch in each diameter; and the edges of the perforation were raised and indurated, giving the appearance of having been punched out of the gut. On one side of the perforation in the same patch, but separated by a very narrow band of tissue, was a second small perforation communicating with the abdominal cavity, however, through the same opening into the peritoneum as the larger perforation. On the outside of the gut the peritoneum was stripped up from the muscular layer back and all around the edge of the perforation. Numerous ulcerations of Peyer's patches were observed,

and one about two inches by one inch in area was of great thickness and the seat of several small sloughs. The peritoneal surface over this patch was much inflamed and was covered with much lymph. The kidneys were rather large and soft, the capsule adherent, but the pyramidal and cortical portions had normal relations. The spleen was three times its normal size, and quite soft and friable.

Dr. Fussell said: This case seems instructive from several points of view:

1. Aside from the disease which most interests us, and which was the direct cause of the patient's death, he had an old peri-typhlitis. This was probably not due to any disease of the appendix; at any rate there was no destructive disease of this part, although in the absence of a microscopic examination it is impossible to say there was no catarrhal inflammation. This peri-typhilitis had become well spontaneously, and there was no sign of pus in the neighborhood. The symptoms to which this attack gave rise were those of *dysentery*, not an unusual thing, I am persuaded, where there is inflammatory disease in the cæcal region.

2. The absence of diarrhœa to any extent (six stools in four and a half days), and the existence of such grave intestinal lesions, is additional proof that it is impossible to diagnose the amount of intestinal involvement from the severity of the diarrhœa.

3. This case shows the uncertainty of prognosis in typhoid fever, especially where the disease has not been vigorously dealt with from the beginning. This patient entered the hospital with a temperature of 105° F., and gradually the latter fell until it was marked as 101.5° F. There was no diarrhœa, and there were no abdominal symptoms. Although the pulse was never good it was not alarmingly bad. Indeed, from such a complex of symptoms, I was tempted to give a most favorable prognosis before the sudden and fatal collapse.

4. This case, although proving nothing in itself, is one of a series I hope soon to present before this Society, in which salol had an apparently good effect upon the course of the typhoid fever.

In answer to a question from Dr. M. O'Hara, Sr., Dr. Fussell stated that in this case there had been practically no hemorrhage from the perforation. Dr. Formad remarked that in a number of cases brought to his notice there were many in which no hemorrhage had occurred. He had often met with walking cases of typhoid fever and pneumonia, walking about with the gravest lesions of the disease; but did not recall to have met with such cases existing any time after the occurrence of perforation. *September* 24, 1891.

3. *Hemorrhagic erosions of stomach.*

Presented by Dr. Henry W. Cattell.

The individual from whom this specimen was obtained was admitted to the Philadelphia Hospital in a nearly starved condition, and died shortly afterward from phthisis.

The specimen, the entire stomach of the man, presented on the mucous surface several small, rounded, and irregular excavations, showing only as roughenings for the most part.

"The only three things that these slight excavations could be are hemorrhagic erosions, post-mortem digestion of the membrane, or tubercular infiltrations with secondary erosion. Dr. Formad diagnosed the specimen as one of hemorrhagic erosion, and as he considered it a fine example of the condition, I present it before the Society. The erosion is not as well marked since the specimen has been in the alcohol as it was in a fresh condition."

Dr. Formad remarked the greater rarity of these hemorrhagic erosions than that of fully formed gastric ulcers.

Dr. J. L. Hatch asked: Were not these erosions a cause of gastric ulcer?

Dr. Formad replied that they are not. The ulcers are the result of embolism, while here multiple hemorrhages occur all over the stomach.

Dr. Cattell stated that the condition is to be regarded as due to the malnutrition of the part; and pointed out that healing of such erosions is more easy of accomplishment than in case of gastric ulcer.

September 24, 1891.

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4. *A case of giant growth of the colon, causing coprostasis, or habitual constipation.*

Exhibited by Dr. Henry F. Formad.

The case to be recorded, and from which I present the specimens, is unique in the history of morbid anatomy. It represents a human colon of dimensions equal to that of a cow, and the accompanying illustration, which is a reproduction of a photograph taken side by side and simultaneously with a normal human colon, will convey to those who did not see the specimen an adequate idea as to its size. (See Fig. 3.)

The exact measurements of the different parts of the bowel will be given hereinafter.

Definition. The specimen is one of *giant growth* of the colon, or a congenital anomaly belonging under the category of *monster per excessum*, followed by coprostasis or habitual constipation, which eventually terminated fatally in the adult. This case differs from all on record in the following points:

1. In that the enlargement or dilatation preceded and was the cause, and not the result, of coprostasis.

2. There was no acquired morbid change, such as colitis or obstruction of the bowel, that led to this condition.

3. The mode of death was not due to ulceration or perforation of the bowel, or to peritonitis, as in the cases on record.

4. The specimen exceeds in magnitude the few cases heretofore recorded.

History of the case. J. W., aged 29 years, white, single, was found dead in the water-closet on the premises of a society to which he belonged, and the case subsequently became subject to the investigation of the coroner. Being delegated to make an autopsy and an investigation of this case, my inquiries revealed the following facts:

His mother tells that up to the age of one and a half years the subject under consideration was a normal infant, with the exception of a rather large abdomen, frequent irregularity of the bowels, and attacks of constipation, but no other deformity of the body had been noticed by her up to that time. Subsequently, and especially noticeable at the age of two years, the abdomen began to swell and the disturbances in defecation began to be more marked, so that constipation would last from two to four days as a rule. His appearance was said to be that of a marasmic child, lean and emaciated, and until five years of age he was unable to rise without assistance. Subsequently the bodily development was progressive, although he was rather spare. At the age of 12 years he was able to go to school, and although the history of his intellectual success as a schoolboy is uncertain, he appeared to have had the normal intelligence of lads of his age. At the age of 16 years he earned his living at a foundry, for eighteen months, at continuous work. Subsequently he worked for several years as a laborer at an oil refinery, and, while his work was uninterrupted during this period, his parents say that he was subject to habitual constipation, said to last as long as a whole month at a time, although no correct clinical data covering this period of his life could be obtained. Yet it was obvious that his abdomen continued to grow in size. At this period

of his life, while not feeling distressed by any painful ailment, he is said to have visited the dispensaries of various hospitals of this city. At 20 years of age his abdomen had reached very large dimensions, and the figure of his body became so peculiar that the managers of the Ninth and Arch Street Museum saw fit to put him on exhibition as a

FIG. 1.

Case of coprostasis, showing distention of abdomen. Front view.

"freak," and for eight or ten years he was known as the "wind-bag" or "balloon-man." It is said that the circumference of his body at this time was equal to his height, and that he was able to diminish or increase the circumference of his abdomen and chest at will to the extent of fourteen inches. He took particular delight in pounding himself upon the abdomen with his fist, and permitted spectators at the

Museum to do likewise. Eye-witnesses say that when his abdomen was thus pounded it resounded like a drum. It appears evident that his abdomen must have been larger at previous periods of his life than at the time of death. For about one year he ceased appearing as a museum freak, and acted as a messenger for a market, being quite a well-known figure on the streets during the year previous to his death.

Fig. 2.

Case of coprostasis, showing distention and shape of trunk. Side view. [All three illustrations relate to the same case.]

The whole history of his life and habits does not present anything peculiar, except that he had an enormous appetite and was generally a good feeder. He was known to relish a few extra heavy meals a day. He was occasionally of intemperate habits.

He applied lately to the Hahnemann Homœopathic Hospital, complaining of obstinate constipation of his bowels, general weakness, pain in the abdomen, shortness of breath, and great tympanites. He complained also of having a passage of his bowels only once every few weeks. He was admitted as a patient under the care of Dr. Vischer, one of the attending surgeons of the hospital, being admitted to the surgical wards under the supposition that there was a large abdominal tumor or some obstruction of the bowels that had led to the enormous distention of the abdomen. He was etherized, and, to the credit of Dr. Vischer, it must be said that a simple enlargement of the colon, without any other complication or tumor formation, was made out. It is evident that the case was carefully studied by the physicians of the Hahnemann Hospital, and the two accompanying reproductions of the photographs—one showing the front and the other showing the side view of the patient—were obtained through the courtesy of Dr. Vischer. These photographs convey most excellent ideas of the physical condition of the patient, especially as they were taken within a short time of his death.

The patient was dismissed from the hospital, but frequented for some time the dispensary of the same until the day of his death, when he was found dead in the water-closet of a restaurant, as stated.

Autopsy, twenty-four hours after death. Body of very peculiar configuration. While the abdomen and chest were enormously distended, the body was that of a very lean subject. Bodily height, 5 feet 7½ inches; total body weight, approximately, 150 pounds. Circumference of abdomen on line of umbilicus, 7 feet 2½ inches; circumference of chest below nipples, 5 feet and a fraction of an inch. (Figs. 1 and 2 give a quite adequate appearance of the shape of the body, yet the dimensions of the thoracic and abdominal cavities were greatly augmented at the time of death, or rather at the autopsy.)

The skin had a glossy, pale appearance, and about the abdomen there were some post-mortem discolorations of the integument, most marked in a broad line about five inches wide, going obliquely from the right hypochondriac region toward the left groin. The abdomen was extended by gas, like a drum, and a large quantity escaped on incision. Section of the integuments of the body revealed nearly complete absence of the *panniculus adiposus.* In fact, there appeared to be general absence of adipose tissues, not only in the trunk of the body, but also in the integuments of the extremities, there being even less in the latter. There was also a general atrophy of the muscles of the body. The extremities were reduced to about two-thirds of their normal circumference.

Fig. 3.

Human colon, congenital giant growth and coprostasis. The more distended end is the sigmoid flexure. The narrow part taking exit from it represents the greater part of the rectum, which was normal. The narrow distal end of the preparation represents the head of the colon with the string attached to a fragment of the small intestines. The arched part of the specimen represents the transverse portion of the colon. The figure within represents a normal human colon photographed simultaneously for comparison of dimensions. Dried preparations.

The peculiar disfigurement of the body was found to be due to an enormous distention of the abdominal cavity, with huge increase of its viscera, encroaching upon the thoracic cavities. The spine was unduly straight. The ribs were normal in number, but anomalous as to direction of their growth. They all extended at right angles from the axis of the spine, which, as stated, was remarkably straight. In other words, the ribs—even the lower ones—pointed upward instead of downward; and the sternum, which was unusually short, measuring only 4½ inches, pointed at an angle of 90 degrees from the cervical portion of the spine.

The distention of the peritoneal cavity had pushed up the diaphragm as high as the third intercostal space anteriorly and the fifth dorsal vertebra posteriorly. The thoracic cavity was reduced to about one-third of its normal capacity. The heart was normal in every respect, but occupied a perfectly horizontal position to the axis of the body, the apex occupying about the second intercostal space to the left side of the sternum.

The section of the heart and aorta did not reveal anything abnormal, all the valves and orifices being perfectly consistent, and the musculature of the walls perfectly proportionate.

The blood was absolutely fluid in the cavities of the heart, as well as throughout the rest of the body, indicating that death must have been instantaneous. The pleural sacs contained no fluid, and, like the pericardium, showed nothing abnormal except diminution in size. The lungs likewise occupied a horizontal position, the apices pointing toward the spine, whereas the basis of these organs faced the sternum lying between the first and third ribs. Sections of the lungs showed them to be highly congested from passive hyperæmia and somewhat œdematous; otherwise the parenchyma of the organs was normal.

Abdomen. No excess of peritoneal fluid, although the surfaces of the peritoneum had an unusual degree of moisture. The color of the surfaces was normal, no evidence of any hyperæmia or inflammatory conditions in any part. A most striking appearance was presented by the colon. It was distended by fecal contents and by gas, and although occupying a normal direction in the abdomen, it was of huge dimensions and occupied large portions of the thoracic regions of the body. By a rough estimate it had the appearance of being at least ten times wider than normal, the exact measurements being as follows: Total length of colon, 2.52 metres (about 8 feet 4 inches). The rest of the figures relate to the circumference of the bowels: Cæcum, 26 c.m. (10 inches); colon, ascending part, 37 c.m. (15 inches); colon, transverse part, gradually

increasing from 38 c.m. to 76 c.m. (15 inches to 30 inches); colon, descending part, 60 to 62 c.m. (24 to 25 inches); sigmoid flexure, 62 to 69 c.m. (25 to 27 inches).

The meso-colon was abnormally large and thick, which, however, was perfectly consistent with the enormous hypertrophy of the colon.

The whole of the colon thus presented a gradual increase in size or width from the cæcum to the sigmoid flexure, the greatest increase in width being in the transverse portion.

There were no distinct angles or M-shaped figuration of the colon or the so-called kinking of the bowel, as has been described in some cases. In fact, no mechanical, visible, or obvious reason for obstruction could be discovered in any part of the tube.

The contents of the colon was represented by two and a half pailfuls of feces, which weighed forty pounds. The physical character of the feces appeared to be normal, appearing as a semi-fluid, dark-brown mass, with perhaps a somewhat greenish tinge; microscopically and chemically nothing abnormal was discovered.

The rectum was perfectly normal in dimensions; its muscular coats were quite thick, and it presented a striking transition from extreme dilatation of the sigmoid flexure and the rest of the colon above to that of contraction, although no abnormal anatomical appearanees of contraction that could have led to any obstruction could be discovered either in the rectum or anus.

This remarkable excess in size was, however, limited to the large intestine; the small intestines, as well as the rest of the alimentary canal, being of normal dimensions. The vermiform appendix also was normal, measuring 15 c.m. (6 inches) in length and 1 c.m. ($\frac{2}{5}$ inch) in width. It was patulous, and contained a small amount of fecal concretions.

The coats or walls of this specimen of large intestine, even when examined fresh, did not appear to be unduly thick; in fact, the bowel presented to the naked eye no alterations except those referring to the muscular coat, which will be described below. There was a notable absence of sacculations throughout the whole of the colon, evidently due to an abnormality of the muscular coat and a remarkable lack of epiploic appendages. I have stated before that there was a nearly total absence of visible adipose tissue, and this particularly holds good for the peritoneum and omentum. The omentum was ill-developed, and was found retracted or rolled up toward the diaphragm. The whole alimentary canal, with contents intact, was removed, ligatures being put at the œsophagus and rectum, and, without the liver, proved to

present the actual weight of 57½ pounds, and although, unfortunately the colon was not weighed by itself, at least 47 pounds must be allowed for its weight, for the stomach and the small intestines, being normal in size, hardly represented a weight exceeding 10 pounds, as will be seen below.

Stomach. Contained some undigested food, which presented the appearance that death had ensued after a full meal. The stomach was of absolutely normal dimensions, and showed no other pathological changes than some thickening of the mucosa with mamillation or folds. Microscopical sections revealed a simple hypertrophic condition and a catarrhal condition of the gastric and peptic glands, and a decided increase of connective tissue in the submucosa. The muscular coats were also somewhat increased in thickness. The œsophagus was normal. Duodenum and small intestines did not present anything visibly abnormal, either as to length or calibre, and microscopic examination made from the fresh specimen showed nothing beyond some slight catarrhal change, and some increase in the volume of the tissues in both the submucosa and muscular coats.

Liver. Weighed 3½ pounds. Shape of the organ somewhat deformed, the left lobe being nearly as large as the right. There was nothing otherwise noteworthy. Its color, consistence, and surface presented nothing abnormal.

Gall-bladder and contents normal, and ducts likewise.

Pancreas and duct normal, there being only a few hemorrhagic foci in the glandular structure. The only abnormality, which relates also to the liver, was that of position, both organs being located in a region normally belonging to the thoracic cavity, approximately as high as the fourth or fifth rib or intercostal space.

Spleen. Weighed six ounces. Normal in appearance, but in a position at least six inches higher than normal, and unusually movable and without any contact or proximity with the left kidney.

Kidneys. Normal; also supra-renal bodies.

The *semilunar ganglion* and a few other nerve plexures were secured for microscopical examination, the result of which was rather negative, because nothing abnormal could be seen, except, perhaps, some undue pigmentation of the ganglionic cells, a condition that is sometimes observed in perfectly normal cases.

Urinary bladder. Ureters and generative organs perfectly normal, except that the left testis, which was larger than the right, showed a few small cysts.

Brain showed nothing abnormal.

Cord, not examined.

A close search was made for any evidences of obstruction of the bowel, of recent or old origin, but in no part of the intestinal tract was any lesion discovered that could or would have led to obstruction.

The great hypertrophy of the colon thus finds no explanation in any morbid or acquired anatomical lesion. Examination of the walls of the large intestine failed to reveal any morbid changes whatsoever beyond a numerical hypertrophy.

I much regret that the examination was not made at a time when the specimen was fresh, on account of our anxiety to preserve the specimen unmutilated, and to enable us to make a dry preparation after the specimen was inflated to exactly the same proportions as found at the autopsy. Only subsequent to this, and after the specimen was fairly dried, a microscopical examination was made of three parts of the colon, viz.: ileo-cæcal valve, transverse colon, and near the rectum. Although the examination of these specimens, which had to be remoistened, did not yield quite satisfactory microscopical sections, yet they presented the same appearances as a normal colon did prepared under the same circumstances and conditions.

The naked-eye appearance of the specimen when fresh failed to indicate or suggest other than structural change, those of bulk or size; in fact, the walls of this colon, if anything, are somewhat thinner than normal.

From a morphological standpoint it may be said, however, that besides the giant growth, this colon presented an abnormality in the distribution of the longitudinal bands of muscular tissue, and possibly also of the circular coat. The well-known three bands, or *tæniæ*, that traverse the colon longitudinally were unusually ill defined, in fact, appear to be missing in certain parts of this bowel, or are so uniformly dispersed that they have lost their individuality.

The lack of the prominence and effects of these muscular bands easily explains the rather imperfect sacculation of the walls of this colon.

From the above it is evident that we deal here with a uniform hypertrophy of the colon, which might be defined as a numerical hypertrophy without morbid change or morbid cause, except those of excess in bulk or size, and we must class this specimen as one belonging in the category of giant growths or *monster per excessum*.

Literature. An examination of the literature of this subject, both as regards the congenital hypertrophy or dilatation, or constipation leading to dilatation, revealed but a few cases in which the enlarge-

ment of the colon had reached such dimensions as in the case under consideration; and, in fact, no case in all its bearings parallel to this was found.

The cases of congenital anomalies recorded refer to large diverticulæ of the colon. The most interesting of these was described by Grawitz,[1] in 1876, in which a large diverticulum formed the contents of a hernial sac, the rest of the colon presenting only a moderate dilatation.

2. The case of Gruber[2] represents a case of an enlarged sigmoid flexure, which measured from 10 to 13 c.m. (4 to 5 inches) in diameter; whereas, the rest of the colon showed only moderate dilatation, its total length being 1.32 metres (52 inches). There was displacement of the sigmoid flexure to the upper part of the abdomen and very marked chronic peritonitis, adhesive bands having been instrumental in dragging the flexure from its normal position.

3. Little and Gallaway[3] describe the case of an imbecile, male, aged thirty-four years, 6 feet in height, whose abdominal measure in the region of the umbilicus was 45 inches around, and who was admitted to the hospital on account of long-standing obstipation.

He died seven days after admission. The autopsy showed the transverse colon to be 6 inches in diameter (about 19 in circumference), while the descending and sigmoid portion measured 12 to 20 inches in diameter. There was decided colitis and thickeniug of the mucous membrane, and numerous ulcerations.

4. Peacock[4] records a case of a male, aged twenty-eight years, teacher of vocal music, who had suffered from constipation since birth.

He frequently resorted to enemata, without which there would be no evacuation of feces, but much flatus, which necessitated frequently his leaving the room to expel wind. He enjoyed good health up to six weeks before death, when he was said to have died from heart-failure.

The autopsy showed that every organ in the body was normal except the colon, which was enormously and uniformly distended, measuring 6 to 8 inches in diameter, and containing fifteen quarts of fecal matter, semi-fluid in consistency and green in color. There was also decided chronic colitis, with extensive ulceration. The rectum was not involved.

5. Lewitt[5] gives an account of an interesting case.

White male, aged twenty-one years, suffered from obstinate consti-

[1] Virchow's Archiv, vol. lxviii, p. 506.
[2] Ibid, 1872, vol. lxii, p. 432.
[3] Transactions of the Pathological Society of London, 1850, vol. iii, p. 107.
[4] Ibid, 1872, vol. xxiii, p. 105.
[5] Chicago Medical Journal, 1867, vol. xxiv, p. 359.

pation that would last eight to ten days at a time for nine years. When he came under medical observation he suffered great agony from pain in the abdomen, with frequent desire to expel flatus, which could only be accomplished by standing on his head and hands to give his body a perpendicular position.

The abdomen was greatly distended and tympanitic. Death resulted from peritonitis. The peritoneal cavity contained fecal matter, due to perforation of the colon in several places. The ascending and descending portions of the colon lay as large columns, side by side, for there was no evidence of a transverse part of the colon. The colon was filled with soft fecal matter, measuring about a pailful. The mucous membrane of the colon was much thickened and ulcerated, which had led to perforation and peritonitis, as stated.

The measurements of the colon are given as 5½ inches in diameter in both its ascending and descending parts. The accumulation of feces appears to have been most marked in the sigmoid flexure, which pressed upon the rectum with such force as to prevent defecation, and by this the author of the paper explains the peculiar position which the patient had to assume to expel flatus or feces.

6. Cruveilhier[1] speaks of a case of an old woman suffering from coprostasis, in which the colon was distended to the size of a horse's colon, and was filled with thick feces.

The same author refers to the case of an old man, affected similarly in whom the colon measured 35 cm. (14 inches) in circumference, the cæcum being the size of a child's head.

There are numerous other cases in the literature, which refer to coprostasis of long duration, lasting as long as seven months; of individuals having a passage of feces only a few times a year, with more or less dilatation of the colon, but in most of which there appears to have been some ulceration of the mucous membrane. But the descriptions of the cases are so deficient, or in some instances so inaccessible, that no use can be made of them. Moreover, they have no direct bearing upon or similarity with my case.

There are, also, some doubtful cases in the older literature in which indefinite coprostasis is spoken of, and in which enormous quantities of feces, weighing as much as sixty pounds, are referred to. Dr. W. W. Johnston, in his excellent article on constipation, refers to such cases.[2]

[1] Referred to by Leichtenstern in Ziemssen's Cyclopædia of the Practice of Medicine, vol. vii, p. 607.

[2] See Pepper's System of Medicine, vol. ii.

Leichtenstern[1] speaks of coprostasis conditioned upon habitual constipation which he terms *ileus paralyticus* in the small intestines, parallel with coprostasis of the large. He attributes this condition solely to the insufficiency of the forces destined to move the contents of the intestines forward, want of peristalsis, and ultimate paralysis of the bowel, which leads to arrest of the passage of feces. This, with insufficiency of abdominal pressure, leads, in turn, to dilatation of the bowel.

Undue sacculation of the colon, due to contraction of the longitudinal muscular coat or the failure of the proper development of these muscular bands, and likewise an abnormal meso-colon, are considered important factors of coprostasis and subsequent dilatation of colon.

I am indebted to Dr. R. P. Harris for the following note, which has some interest in illustration of the cases on record, but does not correspond in all its features with the case under consideration.

"*Fatal constipation from neglect* is the most apt to occur in lunatics living at home, where their habits are not closely looked into, and the measure of their discharges is not known by those who reside with them. One such case came under my observation in 1848, in a single lady, thirty-eight years of age, who had been the subject of melancholia for twelve years. She had been in the habit of visiting a yard-privy by herself, and it was not known to her parents that she did not evacuate any fecal matter. When taken sick to bed she had frequent peristaltic pains, like the early contractions of labor; passed small quantities of softened fecal matter; was affected with hæmorrhoids, and her urine dribbled from her. From her caput coli to her anus her large intestine was packed with solid fecal matter which could not be removed, and she died in five days after I was called in.

"An autopsy showed that her rectum was so distended that it filled the pelvic cavity, pressed the uterus against the pubic bones, the organ being white and bloodless, and prevented the filling of the bladder. The fecal matter in the rectum was as hard as brick clay is when ready for the mould, and the whole of the large intestine was distended to a diameter varying from three to four inches. She must have been constipated for a number of weeks."

Résumé. A congenitally large tube, like the colon in this case, with anomalous arrangement of the muscular tissue, is naturally unfit for contraction or any necessary physiological exercise, probably for want of proper innervation; in other words, the colon presenting a large cavity

[1] Ziemssen's Cyclopædia of the Practice of Medicine, 1876, vol. vii, p. 583, American publication.

whose boundaries were not under physiological control, it is perfectly natural that the regular expulsion of feces would be abnormal.

It is natural to suppose that, under such circumsiances, it would take a great accumulation of feces to fill the cavity, and that such accumution would take an abnormal time to be expelled. It might take many days before the accumulation would be sufficient in bulk to make an impression that would promote a physiological contraction sufficient to expel it. It is thus plausible that an evacuation of the bowels might be deferred for weeks, or even a month, as in the case under consideration.

It is evident, from the study of this case, that we deal here primarily with a congenital anomally, due to an anomalous arrangement of the muscular fibres of the longitudinal as well as circular coats of the colon. Contrary to the usual rule, where constipation or coprostasis would cause a dilatation of the walls of the gut, a dilatation of the gut congenitally malformed and distended, was the factor in causing the coprostasis or the accumulation of feces in this case. The absence of sacculations and folds, which underlie the physiological cause for the accumulation and hardening of feces, was also wanting in this case, and thus the normal stimulus upon the mucous membrane of the colon was missing.

Whereas, there was no evident histological evidence of any change in the nervous structures in this case, it appears to me probable that the want of proper innervation of the colon—in other words a congenital paralysis of this bowel—was a very important factor in producing the dilatation described.

I wish to acknowledge the aid, in the study of this case, of my assistant, Dr. J. L. Wetherell, who assisted me at the rather voluminous autopsy and the subsequent study of the case—a work which consumed a great deal of time. *October* 22, 1891.

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5. *Gunshot wound of mouth.*

Exhibited by Dr. H. F. Formad.

These specimens were removed at the autopsy on a man, aged 72 years, who committed suicide by shooting himself in the mouth. The tongue was badly injured, and the bullet lodged in the body of the third cervical vertebra. There was very little hemorrhage.

The kidneys are of the red granular variety, but without marked contraction.

Dr. G. G. Davis: The lack of hemorrhage is to be explained by the

fact that the path of the ball lay along the middle line, where there is less danger of injury to arteries than on either side. *November* 12, 1891.

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6. *A case of colloid cancer of the stomach terminated by gastric perforation; autopsy.*

Exhibited by Dr. E. C. Ellett.

Cancers in young people, *i. e.*, under 40 years of age, while not rare, are unusual enough to render their occurrence noteworthy. The colloid variety is a rare form of cancer, so rare that there is but one specimen in the entire Pathological Museum of the University of Pennsylvania. A case of colloid cancer occurring in a young subject must, then, be worthy of record. Such a case is the following, which also presents difficulties in diagnosis, a rather short course and a peculiar termination.

I am indebted to Dr. John K. Mitchell for his kindness in allowing me to report the case, which occurred during his term of service in the medical wards of St. Agnes' Hospital.

J. H., aged 33 years, male, white, single, a native of Belgium, and a metal-worker by trade, was admitted to Dr. Mitchell's ward, in St. Agnes' Hospital, on the 27th of August, 1891, with the following history:

His father is living and well. His mother is dead, of a cause unknown to the patient. There is no history of cancer in the family. The patient has always been healthy, and is not a drinking man. He has been in this country since April, 1891.

He has been sick since May, 1891, with vomiting, pain in the abdomen and constipation. He always vomits after eating, sometimes immediately, sometimes after a little interval. Usually the ejecta consist of the food recently taken, but he has vomited blood twice. Fruit and milk always make him vomit; sometimes he retains meat. He has pain in the epigastrium after eating, no pain when the stomach is empty. The pain is burning in character, and increased by pressure. Constipation is the rule, but is not obstinate, and for the last three days he has had one stool a day. He has lost much flesh and strength since his illness began. His appetite is good. He has had no chill or fever. The tongue is moist and slightly coated. The heart and lungs are normal. The belly is markedly scaphoid. There is no enlargement of

the liver or spleen. There is no tumor palpable in the abdomen. The patient is emaciated, and the skin has a dirty-yellow color, with a constant dull-red flush of the face. The urine is normal. Temperature and pulse-rate are normal.

Gastric ulcer was suspected from these symptoms, and the diet was restricted to milk and lime-water, the patient put to bed and a pill of one-eighth of grain of nitrate of silver ordered thrice daily by Dr. C. W. Burr, who had charge in Dr. Mitchell's absence. For a while, under this treatment, the patient improved. For four days he did not vomit, then he vomited every other day for a week, always after taking food or medicine, especially if taken in considerable quantity. The temperature was not elevated, nor did the pulse-rate deviate from the normal. The bowels were irregular, frequently requiring laxatives, and not rarely astringents for diarrhœa, which was always easily controlled. About a week after admission his tongue gradually assumed a heavy black coat, especially marked in the centre. The substance composing it was of considerable firmness and tightly adherent to the mucous surface, so that the latter bled when the coating was stripped off. A portion of this substance under the microscope showed a homogeneous black mass, bearing no resemblance to any familiar object. Possibly it was a deposit of oxide of silver, although that is not probable, as he was taking silver in pill form, and presented no signs of argyria. This coating disappeared slowly in about three weeks, and the tongue assumed its former fairly healthy appearance, which it maintained.

The patient did not thrive on milk and lime-water, and despite the vomiting his diet was made more extensive. The dyspeptic symptoms remained only moderate, and he was soon allowed the ordinary "house diet," but the vomiting was so promptly increased that the diet was again restricted to milk, and later this was peptonized. Cascara sagrada was ordered three times daily for constipation, and his stomach was washed out in hopes that the vomiting might be relieved. The tube brought away curds of milk. The test for free hydrochloric acid was not applied. Oxalate of cerium was ordered for the vomiting.

Asthenia became more marked, and the dirty-yellow hue of the skin more pronounced. From the progressive and persistent loss of flesh and strength, the cachexia, the constant epigastric pain and dyspeptic symptoms, Dr. Mitchell concluded that there was malignant disease of the stomach in spite of the youth of the patient, his good family history as to malignant disease, the absence of the characteristic "coffee-ground" vomit, and the absence of any appreciable tumor. Accordingly, the nitrate of silver was withheld, and subsequent treatment, both

dietetic and medicinal, was symptomatic. Blisters to the epigastrium proved tolerably effectual in relieving pain.

The history of the case for the next three weeks does not differ much from what has preceded. About the 7th of October the face became swollen, especially the lower eyelids, and this, after two days, disappeared as suddenly as it came. A week later this condition was again present, to disappear as before in two or three days. The urine remained normal. Its quantity was not estimated. The subjective symptoms varied much. Epigastric pain was pretty constant. The patient felt well enough to be out of bed most of the time, nor was he confined strictly to the house. He had an occasional headache. The chest was examined repeatedly and no abnormality noted. No tumor or thickening could ever be made out in the abdomen, and the belly retained its scaphoid shape till the day of death, though later less marked than at first.

On the morning of October 25th examination showed the lower half of the abdomen distended, resonant, and tender. Above the umbilicus it was retracted. He had some pain in the left chest and shoulder. Over that side of the thorax the expiratory sound was prolonged, and a few râles were heard. The percussion note was normal on both sides. Patient said he had passed no urine for eight days—probably a misstatement. The catheter drew half a pint, which examination showed to be normal. He was constipated, and a purgative was ordered. In the afternoon he passed two ounces of urine, but the bowels did not move. On attempting to give an enema the rectum was found full of scybalæ. It was "quarried" out, and the patient felt much relieved. The evening temperature, taken about 5 P.M., was 99.4° F., and at that time he was resting comfortably and quietly. At 7.30 P.M. he was found to be in collapse, with a cold sweat, cold extremities, hands cyanotic and speech incoherent. The temperature was not taken. Stimulation by hypodermatic injection was freely resorted to, together with warm applications externally, but with no avail. He died at 9.45 P.M.

Autopsy fifteen hours after death. Rigor mortis slight. Cachexia marked. Body emaciated.

Thorax. Old pleural adhesions on left side. Effusion of straw-colored serum was present in both pleural sacs. The lung substance was normal. The pericardial sac contained three ounces of straw-colored serum. The heart was rather small, and the veins on its surface were much dilated. The cavities, the valves, and the aorta were normal.

Abdomen. General peritonitis was found, with a moderate amount of recent lymph along the lines of contact of the coils of intestines. A

considerable quantity of straw-colored serum was found in the abdominal cavity, and in it were floating masses resembling curds of milk. The intestines were distended and slate-colored, and covered with the exudate mentioned. The stomach was small, the walls thick and hard, especially toward the orifices. It was of a dull-white color, contrasting strangely with the darker intestines. It was tightly adherent to all neigboring structures, and was removed with difficulty along with the spleen, pancreas, and part of the diaphragm, to all of which it was closely bound. The cardiac and pyloric orifices were rigidly open, and barely admitted the first joint of the index-finger. On opening the organ its walls were found to be universally thickened, especially at the orifices, and were white and gelatinous in appearance. The wall was three-fourths of an inch thick at the cardiac extremity, three-fourths of an inch at the pyloric, and one-half inch throughout the rest of the organ. The lower extremity of the œsophagus was slightly involved, but the growth ceased abruptly at the pyloric orifice, as is the rule with these growths, and the duodenum was perfectly healthy. The interior of the stomach was rough and covered with soft excrescences, somewhat resembling huge œdematous granulations. There was no ulceration except at the fundus, where the stomach wall was thinnest and least involved in the carcinomatous process. Here, on the fundus, slightly posterior to the line of the greater curvature, about half an inch from the diaphragm and midway between it and the upper margin of the gastro-splenic folds of peritoneum, was a circular perforation about the size of a silver dime. Its margins were clean cut, and resembled very much the appearance of an ordinary gastric ulcer. The process had started on the mucous surface, and the shelving of the edges was very slight. It would seem that the perforation had existed some time, being closed by an adhesion to some adjacent structure, the dissolution of which allowed the escape of the gastric contents with fatal result, but no such relation was demonstrable.

The bladder was empty. The other abdominal viscera were normal.

The effusions noted must have occurred almost "in articulo mortis," for no signs of them were present when the patient was examined twelve hours before death.

Dr. Mitchell: The marked cachexia in the case was the only indication of the malignant disease. There was no dilatation of the stomach, and no palpable growth. *December* 10, 1891.

7. *Intestinal obstruction from adhesion of suppurating mesenteric glands.*

Exhibited by Dr. Henry R. Wharton.

Robert C., aged 11 years, was admitted to the Children's Hospital May 7, 1889, suffering with coxalgia and necrosis of the humerus. At the time of his admission to the hospital there was profuse discharge from a wound near the right hip-joint, which had been excised in another hospital, and the patient had an elevated temperature.

On June 4th he complained of abdominal pain, which was followed by vomiting and abdominal distention, and he suffered from obstinate constipation, which was finally relieved, after three days, by frequent enemata. After movement of the bowels the pain and abdominal distention subsided, and he was comfortable until June 10th, when the abdominal pain and distention recurred, and was followed on June 12th by fecal vomiting. No movement of the bowel could be obtained by use of enemata. On June 13th it was decided to operate, as no relief had been afforded to the distention and the bowels had not been opened. An hour before the time set for the operation an enema brought away a large fecal movement, and the symptoms of obstruction rapidly disappeared. From this time the patient was comfortable, and passed the feces freely up to the time of his death, which occurred on June 19th, from exhaustion incident to the general tuberculosis.

At *autopsy* the abdominal organs alone were allowed to be inspected. At a point eight feet above the ileo-cæcal valve the small intestine was firmly bound by adhesions to a mass of suppurating mesenteric glands. Flakes of lymph were seen upon the intestines at many points, and the liver showed marked amyloid degeneration.

This case is one of considerable interest as showing how intestinal obstruction may be brought about in cases of enlarged tubercular abdominal glands. It has recently been the subject of study by Treves, who reports cases exactly similar to this. If this possibility be borne in mind a diagnosis of cause of intestinal obstruction will sometimes be possible when it otherwise might remain obscure. The possibilities of operation are shown by the history of this case, in which the adhesion ruptured of its own accord with relief to the symptoms, the patient afterward dying of general tuberculosis.

Dr. Laplace recalled a similar occurrence in a case of retro-peritoneal sarcoma, and Dr. Formad one of ovarian cyst.

March 10, 1892.

8. *Transfixion of the liver of a chicken by a pin.*

Exhibited by Dr. F. S. Pearce.

The specimen was given me by Dr. Allis, who stated that the liver in its present condition was handed to him by a butcher in Kater Market, who said that his wife was dressing a chicken for the table, and found it and preserved it for her husband. There is no reason to question the accuracy of the statement.

Chickens are known to pick up all sorts of bright objects, and the pin may have been taken up and found its way to the liver.

Dr. Stengel recalled a case reported by Dr. A. J. Smith of an hysterical girl who had swallowed many pins, and at whose death numerous pins were found in the pericardium and even in the heart.

March 10, 1892.

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9. *Cancer of the liver with primary seat in the gall-bladder.*

Exhibited by Dr. C. W. Burr.

C. D., white, female, aged 63 years, died at the Philadelphia Home for Incurables, April 12, 1892.

The following fragmentary notes of her previous history were all that could be obtained: For several years she had complained of pain, sharp and at times stabbing, in the region of the liver, and spreading thence throughout the abdomen. Vomiting occurred but rarely, though more or less nausea and anorexia were present for many months. When first seen by me, two weeks before death, jaundice was absent, and there was no history of its ever having been present. She had been at one time treated for many months for peritonitis.

Condition on admission. Cachexia marked. Patient bed-ridden. Great pain throughout the abdomen, markedly increased on palpation, especially in the region of the liver, so that physical examination is unsatisfactory. There is an area of dulness on the right side in front, extending from the fourth rib to the level of the umbilicus, and passing on the left into the hypogastrium. Nodules are not felt. Slight jaundice. Slight œdema of the ankles. Diagnosis: carcinoma of omentum and liver.

Post-mortem. Examination made eighteen hours after death. Rigor mortis marked. Some œdema of legs. Extreme jaundice.

Left pleural cavity and lung normal. On the right the base of the lung is strongly adherent to the diaphragm, and can only be detached by tearing the lung substance. Lower right lobe is dark red, soft, very slightly crepitant. Throughout the lungs there is marked bronchial inflammation. At the apex there are a few old adhesions. No enlargement of the bronchial glands.

The liver is enormously increased in size. Scattered over the surface of the organ are many light-yellow nodules, varying in size from a pea to a lemon. In many places several have coalesced. There is but little bossing, while umbilication is marked. But little of the liver structure in the right lobe remains uninvolved, while in the left there is quite a great deal of normal tissue. On section the nodules show softening.

The gall-bladder, which is about the size of a small walnut, is the seat of a hard cancerous infiltration, occupying its entire substance. The cavity contains four faceted, dark calculi and about one-half drachm of purulent liquid. In the common duct there is a calculus about the size of a very large pea. Otherwise the ducts are normal. The lymph glands around the lobus spigelia are rather large and quite hard. There are a few soft masses in the peritoneum around the brim of the pelvis.

The heart is normal. The breasts, spleen, stomach, œsophagus, pancreas, kidneys, uterus and its appendages, intestines, and bladder are all normal. There is slight atheroma of the aorta. The omentum is not involved. *April* 14, 1892.

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10. *Abscess of the liver.*

Exhibited by Dr. M. H. Fussell.

William W., aged 45 years, was attacked with what was apparently an ordinary attack of catarrhal dysentery in July, 1891. He recovered quickly, but had frequent relapses, with remarkable loss of strength, for two months. The stools were not examined for amœba coli. At about this period the writer was discharged from attendance on the case. At my last visit I had made a careful physical examination, and aside from a weak heart there were no physical signs.

About one month after, a new attendant was called, and pronounced the case one of phthisis. A third man was consulted, who came to the same conclusion. I was informed that for the last month of his life

there had been rapid loss of strength and flesh, together with much cough and expectoration.

The patient died January, 1892, and I was requested to make the autopsy.

On examination the body was much emaciated. There were no external marks.

On opening the abdomen the edge of the liver was seen to be a hand's breadth below the edge of the ribs. On the upper surface of the part of the left lobe below the ribs was a small abscess. In the convex surface of the right lobe was a large abscess the size of the fist, which had ruptured through the diaphragm. The whole lower lobe of the right lung was in a state of destructive pneumonia, and formed practically a part of the abscess cavity. The colon from the sigmoid flexure to the ileo-cæcal valve was the seat of extensive ulceration. So thin were the walls of the gut that they were often torn through in separating the bowel from its attachments.

In the apex of the right chest was a soft fibroma the size of a fist. This may have been due to a wound received during the war of the rebellion, though no missile was found in its interior.

The pus was carefully examined for amœbæ, but none were found.

April 14, 1892.

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11. *Liver from a case of suspected jungle fever.*

Exhibited by Dr. Henry F. Formad.

The patient from whom the specimen was removed was reported to have suffered with jungle fever, an ill-defined disease of tropical countries. It resembles malaria and certain other diseases, affecting the blood powerfully. The patient in question was deeply jaundiced and greatly emaciated. He had suffered constant diarrhœa for a considerable period before his death.

Autopsy. The most interesting feature was the enlarged liver. This organ weighed twelve pounds; it was evidently cirrhotic, dense bands of connective tissue being apparent in its structure. There was also considerable fatty infiltration, and in this respect the present case differed entirely from a previous one which came under the speaker's observation. In the present case the liver presents the appearance of ordinary hypertrophic cirrhosis. The spleen was also much enlarged. The stomach presented the lesions of atrophic gastritis.

May 12, 1892.

12. *Stomach from a case of hydrocyanic acid poisoning.*

Exhibited by Dr. Henry F. Formad.

The stomach is intensely engorged on the mucous surface, and the appearance would indicate that the congestion extended deeply. The suddenness of death in these cases would seem to preclude great congestion, but in this instance there was proof that some interval had elapsed between the taking of the drug and the death of the patient. The man, a jeweler, was found on the second floor and the poison in his shop below. *May* 12, 1892.

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13. *Traumatic stricture of the œsophagus of a horse.*

Exhibited by Dr. M. H. Fussell.

The horse from which the specimen was removed was eating apples, when one became lodged in the œsophagus. A broom-handle was used to force the apple down and caused a copious hemorrhage. The horse lived apparently well for a long time, but died suddenly. At the post-mortem examination a great mass was found surrounding and invading the œsophagus. The mucous surface was ulcerated and cicatrization was beginning in places. In some portions the muscular coat was exposed. Microscopic examination showed the mass to consist of simple inflammatory tissue. *May* 12, 1892.

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14. *Colloid cancer of the stomach.*

Exhibited by Dr. J. K. Mitchell.

I was called to see the patient from whom the specimen submitted was taken, on April 1, 1892. I found a man aged 72 years, with no special cachexia, a cheerful countenance, and bright eyes, but emaciated to the last point. He was weak but not excessively feeble, and not confined to bed. His history was briefly as follows: He had always been unusually healthy, an active, busy man of affairs, now for several years out of business. In June last he began to notice a loss of appetite, which grew into a strong distaste for food. He suffered no pain from

food, unless a large quantity was taken, when he had a great sense of fulness and oppression. He ate for some months scarcely enough to keep him alive.

He had been, up to the date when I saw him, at various times better and worse, but never able to eat normally. He had had no vomiting, though sometimes slightly nauseated. He drank nothing, preferred semi-solid food or solid, in the smallest quantities, and said liquids especially disagreed with him. On examination the abdomen was found scaphoid, so markedly that the walls lay against the spinal column, which could be readily grasped by the hand. No abnormality of the intestines could be found, though it was easy to examine them very minutely. There was no tenderness anywhere to pressure or percussion. The stomach could not be touched below the xiphoid, nor could percussion elicit gastric tympany below the costo-xiphoid angle. The liver outline was in every direction a little within the normal limits. The tongue was rather dry, slightly fissured, and not much coated. There was no jaundice. After six days of treatment, which need not be detailed, the patient gradually failed and died unconscious on April 6th.

At the *autopsy*, which I was obliged to perform alone and somewhat hastily, I found in the general appearance of the abdominal cavity, on opening it, nothing unusual, except that the transverse colon lay unusually high, but preserving its relations, and the stomach was at first entirely invisible. It was greatly retracted upward, small, firm, and removed with some difficulty, owing to its position and the fact that by request of the family I refrained from section of the ribs or elevating the sternum. Nor was I permitted to remove any other organ, which I regret, as it precludes any certainty as to the primary seat of the growth.

The organ on being opened presented considerable thickening and mamillation of the mucosa, and the walls were decidedly thickened, the submucous layer being of a gelatinous nature and fully a quarter of an inch thick. The disease process was spread uniformly throughout the body of the stomach, and the orifices were free. It was evidently an illustration of that form of carcinoma known as colloid.

Colloid is a form of cancer presenting no recognizable symptoms during life. This may be one of the reasons for its being considered (as Brinton says) the rarest form of gastric cancer. A painless course is rare, but not unknown. The same authority states that 8 per cent. of recognized cases are painless, and this is especially true where, as here, it affects an old person, and does not invade the gastric orifices. In such cases all the symptoms are less marked, though vomiting may be persistent even then, as in the case reported here in February of last

year, by Dr. Ellett from my service at St. Agnes's Hospital, where the lowest portion of the œsophagus was beginning to be invaded by the disease when death took place, about six months after the earliest symptoms, in which vomiting had always been prominent.

I regret that it was not possible during life, owing to the patient's feebleness, to make any examination of the gastric contents or any test for free acid. Although this test is not now looked upon, as for a time it was, as diagnostic of malignant disease, in the absence of acid it is of some value, at least in helping to a diagnosis. *May* 12, 1892.

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15. *Carcinoma of the stomach.*

Exhibited by Dr. E. L. Vansant.

Case I. The patient, a mulatto, aged 45 years, had had typhoid fever and malaria, the latter repeatedly. He had spent some part of his life as a sailor. He was admitted to the hospital five days after the date which he considered the beginning of his illness. There was pain in the right hypochondrium on deep inspiration. The temperature rose in the evenings to 103°, and in the mornings was normal. Quinine at first gave relief, but he soon grew worse, and death came finally from exhaustion. Previously, however, nodules had been felt through the abdominal walls.

The liver was found at autopsy to weigh ten pounds and was studded with nodules. The spleen and mesenteric gland were similarly invaded, and the primary growth was found in the stomach at the pylorus.

Clinically the case was interesting from the apparently rapid course, from the high evening and low morning temperatures, and from the excessive tenderness of the liver, which suggested active peri-hepatitis. A trace of free hydrochloric acid was found in the stomach contents.

Case II. The patient was a man, aged 60 years. His family history was clear, and he himself had been well until five years ago, when he had a bad attack of rheumatism. He had used liquor moderately and denied syphilis. His illness was of nine months' duration. He had dyspepsia and vomiting, but never any blood appeared in the vomit. He lost weight and suffered great gastric pain and distress. After using liquid diet vomiting ceased; but whenever vomiting was provoked the quantity ejected was very great.

Autopsy. There was great dilatation of the stomach, and at the

pylorus a large mass of carcinomatous growth, but this had been pushed up under the liver, and therefore could not be felt. The pyloric orifice was very small, admitting no more than a goose-quill. The liver was highly cirrhotic and presented numerous secondary nodules of cancer.

May 12, 1892.

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16. *Carcinoma of pylorus.*

Exhibited by Dr. Judson Daland.

History not remembered. The stomach showed a large cancerous mass at the pylorus, just above the orifice. Examination of the gut showed that parts of the transverse colon and jejunum were infiltrated with cancerous deposit. Nothing further found with the exception that the infiltration caused adhesions which bound parts of the gut together.

May 26, 1892.

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17. *Cancer of the pylorus with possible gastro-duodenal fistula.*

Exhibited by Dr. E. L. Vansant.

The patient, aged 52 years, was admitted to the Philadelphia Hospital complaining of a tumor in the epigastrium, which had been noticeable for about three years. He had much pain; was unable to sleep or lie on his back on account of shooting pains; but was comfortable in semi-reclining position. Vomited a dark liquid. Much distress in epigastrium. No nourishment taken by mouth, and enemata but partially retained. Died about two months after admission.

Post-mortem. Body emaciated and conjunctiva bile-stained. Thorax showed a caseating upper mesenteric gland. Lungs emphysematous; a calcified nodule, grape-shot in size, in middle lobe of right lung. No infiltration of caseous material on pleural surfaces. Heart showed slightly thickened valves.

Abdomen. Large cancerous mass found at pyloric end of stomach, involving the duodenum. Pyloric orifice not entirely closed. Adhesions found all along stomach and duodenum and possible fistula between the two organs in the middle portion, although this may have been a tear, or due to post-mortem softening, due to the action of the gastric juice. Bile duct pressed upon and adhesion formed between the lower border of the right lobe of the liver and the ascending colon. Anteriorly there

was a cancerous mass with adhesions between the left lobe and the mass in the stomach. Stomach much dilated and filled with about a quart of foul-smelling dark liquid, which exuded from the nose. About a quart of bile-stained fluid in the peritoneal cavity.

Kidneys large, white, and the capsule not adherent.

The question whether there was really a gastro-duodenal fistula, or whether it was a tear, is of interest. From the amount and quantity of the vomiting during life, and still more from the appearance, it seems that this was a tear. Another point of interest is that this appears to be a medullary cancer. The marked degeneration and decay and the softening would indicate the soft cancer, although my experience would show that these are most frequent at the cardiac end. It has extended by contiguity and continuity to the intestines, various glands in the abdomen show the transmission by the lymph vessels and the involvement of the liver, that by the blood. Thus we have one case demonstrating all these modes of transmission: direct extension, lymphatic and hæmatogeneous extension. Again we find here a large white kidney, which I find to be very prevalent in the Philadelphia Hospital in emaciated and broken-down old persons. *May* 26, 1892.

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18. *Duodenal ulcer.*

Exhibited by Dr. Alfred Stengel.

The specimen exhibited was removed post-mortem from a case in which there had been no little difference of opinion as to the nature of the disease. Gastric symptoms had been prominent throughout the disease, and had centered attention on the stomach. The patient, a woman, was about 40 or 45 years old, and much emaciated. There never had been any hemorrhage, but there was pain in the upper part of the abdomen, to the right of the middle line. The immediate cause of death was excessive and uncontrollable hæmatemesis. At autopsy this large ulcer, fully two inches long, was found in the duodenum, and extending upward to the pylorus. There are no features of interest excepting the ulcer itself. *June* 9, 1892.

19. *Primary cirrhotic cancer of the liver.*

Exhibited by Dr. Alfred Stengel.

The specimens, which are portions of the liver only, were removed from the body of a man about 60 years old. The details of the clinical history could not be obtained, but it is known that until about six weeks or two months before death he had been in good health. At first he had an attack of what was regarded as influenza, but he soon grew very ill, and bilious symptoms became marked. The liver was found to be swollen and tender, and there was considerable jaundice. There was no severe pain. At this time the case was regarded as one of catarrhal jaundice, but fever came on, jaundice increased, and the man's general condition became grave, and he died a few days since. It is a matter of regret that a more detailed clinical history could not be obtained, for the lesions discovered are very unusual ones.

Practically the whole interest in this case centers in the liver, for all the other organs were healthy. The liver was enlarged, and slightly irregular on the surface, but the marked peculiarity was irregularly scattered areas of light gray color, in some places thickly studding the surface, in other portions sparse. They were only slightly elevated above the surface. On section through the liver substance, similar areas were discovered within, but more prominently marked were certain dense connective tissue bands or trabeculæ traversing the substance of the organ in various directions. The arrangement of the light areas to the connective tissue bands suggest clusters of grapes upon their stems. The substance proper of the liver was bile-stained, and fatty in appearance. On microscopic examination the infiltrating growth of the liver was found to be carcinoma, and the evident association with cirrhotic tissue at once suggested the title cirrhotic cancer. A rather careful search of the other organs revealed no other primary or secondary new growth. There is little question, therefore, but that this is an example of that interesting form of cancer known as primary cirrhotic cancer, or cancer with cirrhosis of the liver. This form somewhat remotely resembles a perivascular form, in which the infiltration of the organ proceeds from the fissure of the liver inward along the portal vessels. In the form represented by the specimen in hand, the first growth is more frequently in the organ or at its periphery, the situation in which peri-portal cancer last appears.

June 9, 1892.

20. *Cancer of the gall-bladder*

Exhibited by Dr. E. L. Vansant.

Henry B., aged 59 years, a native of Germany, was admitted to the medical ward of the Philadelphia Hospital on May 3, 1892. His family history was good, his father and mother, four brothers and three sisters, being reported as healthy. He had six children, all of whom are said to have been healthy.

He gave a previous history of having had typhoid fever when eleven years old, but since then remained in good health, and has always been able to work until December of 1891, when he began to feel distress in the abdomen, with a sensation of fulness and tenseness. He consumed beer and whiskey moderately during his life; never went on sprees. Denied venereal disease.

Since December, 1891, he lost much flesh and strength. His former weight averaged 145 pounds, but when brought under my care he weighed about 110 pounds. He complained of anorexia, a bitter taste in the mouth, marked constipation, and of stabbing pains in the abdomen when evacuating feces. His sleep was poor, and he had much dyspnœa.

Examination at the time of his admittance to the hospital. External appearance: Patient is much emaciated, cheek-bones very prominent, his skin and conjunctivæ slightly jaundiced. Veins, especially of abdomen, prominent. Muscles wasted. Chest-walls are rigid, and the chest is barrel-shaped. Respirations quick and shallow. Abdomen prominent and almost pendulous. Feet and legs moderately œdematous.

Tongue coated, breath foul, teeth poor. Stools normal in color. Liver dulness much diminished. Dulness and fluctuation over abdomen, showing presence of large amount of ascites.

Respiratory system: All the physical signs of a high degree of vesicular emphysema are present, and the patient experiences much discomfort in the recumbent position. Heart sounds normal but weak. Heart dulness lost from the emphysema. Radials hard and rough. Temporals tortuous and prominent. Urine normal.

The patient remained under my care until the time of his death, on June 14th. During this interval he remained in pretty much the same condition, but gradually grew weaker. He complained at times of some pain in the abdomen, but no particular area of tenderness could be detected. The ascites increased, and, as the dyspnœa became worse, para-

centesis was performed on May 11th, and sixteen ounces of a clear serous fluid removed. This relieved his breathing considerably. His appetite remained poor, the ascites reaccumulated, and on June 12th he was again tapped. This time 210 ounces were removed, the fluid being clear, but slightly bile-tinged, and highly albuminous. He felt easier after the tapping but complained of increasing weakness. June 13th, signs of a right-sided pleurisy were noted, and on June 14th he became gradually weaker and died.

Post-mortem examination: Body of a very emaciated man. Conjunctiva slightly bile-stained. Some post-mortem lividity. No rigor mortis. On the upper portion of the scrotum were eight small elongated subcutaneous nodules, the size of a split-pea, their interiors were filled with a yellowish, cheesy material, which exuded on pressure. A scar was seen in the right iliac region, and a fresh opening midway between the umbilicus and symphysis pubis, where a trocar had been inserted.

Lungs: The right pleural cavity contained two and one quarter quarts of a blood-stained fluid, the pleura being everywhere injected. Recent lymph had been thrown out in one or two places. The right lung was compressed, having had about one-third the normal capacity. Both lungs were very much congested. The left pleura was not effected.

Heart: Extremely small (six ounces), walls firmly contracted in systole. No atheroma. Upon opening the abdomen, the trocar wound was found in good condition. Fluid (ascitic) removed—two and three-quarters quarts. The gall-bladder was much distended, and extended about four inches below the edge of the liver.

Stomach: Showed signs of chronic gastritis, and the intestines evidence of old inflammation. Mucous coats were smooth and covered with a glairy mucus. There were several small black spots on the peritoneal covering of the intestines the size of small peas.

The *spleen and kidneys* were normal. The supra-renals were enlarged and hollow.

Liver: Reduced in size, hard, and cirrhotic, and contained numerous nodules, ranging from pin-head to walnut-size, and situated both on the external surface and internally. Some of those on the external surface were umbilicated, and the interiors when opened contained soft degenerated matter.

Gall-bladder: Enormously distended. At the commencement of duct were two small gall-stones, much thick mucus, and a cancerous nodule which completely blocked the exit. On the inner wall of the gall-bladder there was a soft cancerous growth about the size of a silver quarter. Thirty or forty gall-stones filled the bladder. These were small, about

the size of cherry-stones, crumbled when pressed, and had a dark green nucleus in the centre. The cystic, hepatic, and pancreatic ducts were opened, and found patulous. A small gall-stone was found in the common duct, about one and a half inches from the duodenum, but not closing the duct.

Back of the pylorus, but not involving it, was found a cancerous mass about the size of a goose-egg, arising in lymphatic gland, the head of the pancreas and surrounding tissue.

Microscopical examination confirmed the diagnosis of carcinoma, and showed the growth to be of the soft variety.

Dr. Burr remarked that the interesting question, whether in such cases as this the gall-stones result from cancerous disease of the gall-duct and gall-bladder, or occasion such disease by irritation, has not yet received decisive answer. He had the impression that in the collection of cases of carcinoma of the gall-bladder tabulated by J. H. Musser, gall-stones were found in the great majority, but he did not recall the figures. However, this would be of clinical interest, rather than giving any solution of the etiological questions.

Dr. W. E. Hughes expressed himself in favor of the view that the gall-stones in the majority of cases result from the primary cancerous disease. It is well known that some irritation leading to morbid change in the mucous lining is usually present in cases of gall-stone unassociated with carcinoma; and when cancer is present it is evident the condition necessary to formation of calculi are pre-eminently afforded.

Dr. Vansant closed the discussion with the remark that, though no solution of the exact relation of the two processes might be warranted, the clinical interest of gall-stone associated with carcinoma of the gall-bladder must not be overlooked. Further, as to the primary seat of the process in this case, it was easy to see from the character of the growth on the mucous surface that this was the primary seat, and from the nodular appearance of the lesions in the liver and pancreas, that these were secondary. *June* 23, 1892.

21. *Foreign body found in rectum.*

Exhibited by Dr. Ernest Laplace.

The foreign body was a three-pronged piece of wood, which was found in the rectum of a patient admitted to the Philadelphia Hospital for masturbation. The wood had been in the rectum for about two months. Patient had an attack of dysentery, and while treating this the resident physician in charge found the foreign body, which he took to be a piece of lead-pencil.

Dr. Leidy: Many similar specimens have been found in patients in the Pennsylvania Hospital. One case showed a pencil about seven inches long, which had passed into the sigmoid flexure, perforating the the same, and setting up a peritonitis, from which the patient died.

September 8, 1892.

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22. *Cyst of appendix vermiformis.*

Exhibited by Dr. H. R. Wharton.

Dr. Wharton presented a specimen of an appendix vermiformis containing a cyst. The history of the patient from whom this specimen was removed was as follows:

J. B., aged 43 years, for more than ten years had suffered from more or less pain in the lower part of the abdomen on the right side, which he considered a form of cramp. The pain usually occurred in the morning between ten and twelve o'clock. In May, 1891, he had a severe attack of pain with swelling in the right iliac region, which confined him to his bed for some weeks. He recovered from this attack, but suffered since that time with four similar attacks occurring at intervals. In each of these attacks he was quite ill. After the attacks a mass could be felt in the right iliac fossa for some time.

He was admitted to the University Hospital October 25th, and was operated upon by Prof. Ashhurst on October 29th. On exposing the colon numerous old adhesions were found, and when the appendix was exposed it was found much thickened, and contained a large cyst filled with thick gelatinous mucus, which communicated with the canal of the

appendix. Patient did well after the operation, and is now convalescent.

It is not unusual to find ulcers, foreign bodies, or fecal concretions in the appendix in cases of this nature, but this is the first specimen I have seen which presented a well-marked cyst. The thickened condition of the appendix could be accounted for by the numerous attacks of catarrhal inflammation from which he suffered, and the cyst had probably developed as the result of these attacks.

Dr. Stengel remarked that it was probable that the cyst in this case had the same origin and nature as have mucous polypi in other situations on mucous surfaces; and the fact that we have in this case an outward projection as a cyst would be readily explained by the narrowness of the appendix. *November* 10, 1892.

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23. *Peritonitis, sarcomatous or tubercular, with ascites.*

Exhibited by Dr. M. V. Ball.

A man aged 34 years began to suffer with gradually increasing ascites six months ago, having been perfectly well before. There was a loud musical murmur over the entire heart region. There was no other symptom and no fever. The ascites gradually increased, and was tapped twelve times, the fluid obtained being slightly hemorrhagic the second and third times. There never was any pain, and the patient died of exhaustion after six months. After tapping a lumpy mass could be felt in the abdomen.

Autopsy. The intestines were matted together in several places. The stomach was contracted at the middle, and had an hour-glass shape. The omentum was thick and lumpy. Small masses were found attached to the sigmoid flexure. There was intense acute arteritis of the aorta, and the mitral and aortic valves were thickened. The latter were atheromatous.

No decision had been reached regarding the nature of the disease, but I believe it is sarcomatous.

Dr. Griffith remarked that it would be difficult from the specimens before the society to decide the question of the nature of the disease. The extreme ascites would point to obstruction to the portal circulation. It must, however, be remembered that peritonitis causes similar conditions, though in less degree. The appearance of the specimen is

not unlike that of tubercular peritonitis, but the age of the patient, in the absence of coexisting tuberculosis of the lungs, is directly against this diagnosis. *November* 10, 1892.

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24. *Papillomatous tumors from the rectum.*

Exhibited by Dr. Henry R. Wharton.

These small papillary growths were removed from the rectum of a man about thirty years of age. The whole rectum was studded with growths. The growths here presented have been examined microscopically, and have the appearance of warty tumors, or papillomata.

Mollière describes a granular condition of the mucous membrane of the lower part of the rectum, composed of soft, friable and vascular tissue with great tendency to hemorrhage, occasionally seen in children, under the name "benign fungus of the rectum." There is also a warty condition of the mucous membrane of the rectum associated with stricture, usually syphilitic in origin.

Dr. Stengel remarked that the microscopic features of the growth, as seen in the preparation displayed, showed it to be of adenomatous construction, though a polypoid tumor macroscopically, and he called attention to the tendency of such growths in the rectum to take on carcinomatous characters. This thought is conveyed in the name "benign fungus" given as a distinction from ordinary cases by Mollière, and is well recognized by pathologists. The obvious practical deduction is that early removal gives the best chance for complete cure.

December 8, 1892.

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25. *Cyst, probably dermoid, from floor of mouth.*

Exhibited by Dr. Henry R. Wharton.

I desire to present this cyst removed from the floor of the mouth, which is probably a dermoid.

The patient from whom this cyst was removed was a young man of 25 years, who had noticed a lump under his tongue for about a year. It began to increase much in size since August of this year, and when I saw him, on November 23d, it had pushed the tongue backward and upward to such an extent that it interfered with talking and swallowing.

On November 29th I etherized the patient and removed the cyst from within the mouth without rupturing its capsule. The patient did well after the operation, and was discharged from the hospital on the ninth day.

Cysts of the floor of the mouth are usually mucous or salivary, and are classed under the general name of ranulæ. This cyst is, I think, a dermoid, or, as sometimes called, a sebaceous cyst of the mouth.

The dermoid or sebaceous cyst of the mouth is a congenital cyst, which often remains small and unobserved for years, and then takes an active growth, increasing rapidly in size and interfering with deglutition and talking.

These cysts are caused by the turning in of a small portion of the epiblast, and often contain epidermic débris—a putty-like material, similar to the contents of sebaceous cysts, hair and sometimes teeth.

Dr. Stengel was reminded by this specimen of a case which had come under his notice when a student of medicine. A physician, in operating on a "ranula," found the tumor to contain clear or slightly turbid fluid, and a great quantity of peculiar round bodies, about the size of millet-seeds, and composed of a light-brownish substance resembling cheese in its consistence. There were probably two ounces of them, each one nicely rounded and distinct. On pressing them out and examining microscopically, no definite crystals or other structures were discoverable. Several surgeons and pathologists of experience were consulted as to the nature of the formations, but none was able to give a definite answer. Unfortunately the speaker was not present at the operation, and did not know what appearances the tumor presented. The suspicion of such a cyst as Dr. Wharton presented must, however, be entertained, and seems the most likely condition.

December 8, 1892.

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26. *Tubercular or malignant disease of peritoneum.*

Exhibited by Dr. C. A. Hamann.

The patient from whom the specimens were removed was a man, aged 37 years. He had been ill for some time. At first the disease was regarded as simply appendicitis, and his appendix was removed. Recovery was not complete, and fecal fistulæ were established. One communicated with the exterior through the flank, another with the

bladder, and feces were passed per urethram. After a protracted illness he died of exhaustion.

At autopsy the peritoneal surface was everywhere studded with small nodules, varying in size from a pea to a hickory-nut, flat on the top, and in places somewhat indistinctly dentritic. There was no disease of the lungs or pleuræ, but there were a few nodules on the upper surface of the diaphragm. There was no fluid in the belly. On section the nodules were of uniform consistence, whitish, but nowhere cheesy. Their appearance, therefore, was that of malignant growths rather than tuberculous tumors. *December* 8, 1892.

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27. *Perforating gastric ulcer.*

Exhibited by Dr. J. Hendrie Lloyd.

The patient was a man about 60 years old. Sixteen months before death he complained of dyspepsia, and occasionally vomited after meals. The vomiting would come on with great suddenness, sometimes on the street. There were occasionally radiating pains in the abdomen, but these rarely became marked. Generally there was dull pain, aggravated on pressure. His condition fluctuated. He never vomited blood, but three months ago, after dinner, he had sudden sharp pain, and rapidly sank into collapse. There was no vomiting, but the pain continued, and he became cyanosed. After twenty-seven hours he died.

At autopsy a sharp punched-out ulcer, with a complete perforation of the wall of the stomach, was detected. There was a little hemorrhage into the peritoneum and rapid peritonitis. The ulcer was situated in the lesser curvature. *January* 12, 1893.

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28. *Cylindrical epithelioma of the great omentum, probably originating in the transverse colon.*

Exhibited by Dr. Henry W. Cattell.

J. M., aged 42, white, by occupation a blacksmith, was admitted to the Philadelphia Hospital on August 29, 1892, and died October 5, 1892. Family history good. He had the ordinary diseases of childhood; scarlet fever eleven years ago. About eight years ago was over-

come by heat, and subsequently when exposed to intense heat still suffered from attacks of giddiness. He never had any venereal disease. About eleven weeks before admission he had severe pain in the abdomen in the umbilical region ; also, pain in the back, but recovered to some extent, though he remained very weak. When he attempted to work he would easily become dizzy. For a month before admission he had been having attacks of diarrhœa. At first the stools contained blood, but later, as he describes it, like water. His appetite remained good, but he suffered from nausea after eating. His sleep was much disturbed. His temperature chart shows the highest elevation, 103° F. at 7 P.M. on August 30th, the day after his admission. The chart is very irregular, at times assuming the normal for several days, and then running up to 102° to 102.4°, the temperature reaching subnormal, 96° F., on four successive days preceding his death, and going up to 99° on the evening previous to death. The pulse varied from 120 to 86. The bowel movements were usually frequent, being several days before his death ten to twenty per day. They were loose in character, at times yellow, though more frequently containing clotted blood, and sometimes becoming very dark, due, no doubt, to the presence of blood. The urine contained a small quantity of albumen.

Post-mortem twenty-six hours after death. Body of an emaciated man five feet seven inches in height, weighing about one hundred and thirty pounds. Scars upon middle third of anterior surface of tibia. Post-mortem mottling not marked. A condition of hypospadia, the meatus urinarius opening at the attachment of frenum. The canal continues superficially, admitting a No. 9 sound. Above this opening (6 mm.) is a small false pocket, and continuing to centre of prepuce is a fossa. In the neighborhood of the umbilicus, occupying the entire umbilical region and extending slightly into the adjoining regions, viz., right and left iliac, the hypogastric and the epigastric region, is a rounded mass ; it is raised above the surface and very hard to the touch.

On opening the abdomen a mass extended from the lower border of the right lobe of the liver across the lower border (greater curvature) of the stomach, while the remaining boundaries were formed by the transverse colon, which had been pressed down 5 c.m. below the umbilicus, the tumor thus lying between the greater curvature of the stomach and the displaced transverse colon. From the tip of the xiphoid cartilage to centre of the mass is 14 c.m. ; to the upper edge, 9 c.m. ; to the lower edge, 19½ c.m. The longitudinal distance is 10 c.m. ; the breadth, 11½ c. m. The free edges were surrounded with omental fat and covered by the omentum. The mesentery of the small intestines

was attached to the inner and lower margin, the vessels being numerous, radiating, and deeply stained. The mass was pedunculated, the attachment being to the left of the median line.

The omental attachment at the lower border contained a pocket of yellowish pus. Going internally into the mass to a mother pocket of a larger size, also containing the same character of pus, the outside wall resembled a caseating gland. The contents of the transverse colon were darkish brown (from blood?).

On careful dissection the whole length of the pancreas was followed out from the tail to the head, and there was found to be no involvement of that organ. The position of the tumor might lead one to suppose that it was a tumor of the pancreas. The mass was on both sides of the transverse colon, the new growth was undergoing degeneration, and the walls of the colon were deeply infiltrated. Indeed, it was impossible to separate the mass from the colon without making an opening into the intestines.

The mass on section showed oval and roundish nodules the size of a pea to that of a pigeon's egg, the centre of some of these masses having undergone softening.

The portion of the transverse colon showed on opening dendritic growths standing out in the cavity, the masses being red and the size of pigeon eggs. The liver was markedly enlarged, the lower right border almost extending to the line of the umbilicus. On section it was fatty in character, and weighed seventy-eight ounces. The gall-bladder was distended with a very light-yellowish colored bile. The duct was patulous. The kidneys each weighed five ounces, the capsule was slightly adherent in spots. Two cysts were seen on the surface of the right kidney and one on the surface of the left. The stomach was of a peculiar shape and position, the pyloric end being narrowed like the duodenum, and the cardiac end lying well up under the diaphragm (the left lobe of the enlarged liver helping to push it up), and extending laterally well toward the mid-axillary line. The stomach wall was coated with mucus, the rugæ prominent; the contents liquid. There was nothing of interest in regard to the lungs and heart.

I am indebted to Dr. Joseph McFarland for a microscopic study of a piece from the tumor. He considers it to be a cylindrical epithelioma. The specimen is now under the microscope for your inspection.

The case is interesting from the fact that I am confident if the feces had been carefully examined, small portions of the tumor could no doubt have been discovered, and in all probability diagnosed. The nearness of the papillomatous growths to the rectum would permit of

such pieces being discharged in the feces quickly and without much change, as diarrhœa was usually present.

Dr. W. E. Hughes remarked that the case had presented some of the clinical features of a retro-peritoneal sarcoma, and when the man first came into the wards he was disposed to regard the case as such. Subsequently, however, the tumor began to pulsate distinctly, and he was then led to suspect a deep-seated aneurism.

Dr. McFarland had studied the microscopical appearances very carefully, and found the growth to be a cylindrical epithelioma. He was inclined to regard it as of primarily pancreatic origin from the microscopical appearances. *January* 12, 1893.

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29. *A case of intestinal tuberculosis with sub-peritoneal hernia of omentum.*

Exhibited by Dr. Andrew J. Downes.

Miss K., aged nineteen years, went to work in a mill, September 1891. Her employment was such that sitting before a table she pulled heavy cloth in a manner to strike her abdomen over the gastric region every four or five minutes during the day. She gradually began to complain of pain in this region, she suffered from indigestion and distention of abdomen. During May, 1892, she was obliged to quit this position, and was home sick with a fever characterized by high afternoon temperature. In June a worse attack, with high fever, delirium, and diarrhœa, occurred. She rallied enough to go to New England in August, but caught cold on the way, and fever and diarrhœa returned. On her return to Philadelphia, in October, the case fell into my hands. I found an emaciated, hectic patient, apparently in the last stages of tuberculosis. The lungs showed a very slight deposit in right apex, not yet softened. Cough slight; at times none; little or no expectoration.

Her only complaint was very severe pain in the epigastrium, colic, and at times diarrhœa, loose, offensive, and painful. On examining the abdomen I detected a circumscribed area of dulness midway between the ensiform cartilage and the umbilicus. Under treatment for six weeks; there was decided benefit to pain, colic and diarrhœa. The localized area above referred to remained, although less sensitive. General decline continued, however, and at last I was allowed to explore.

December 20, 1892, she entered a private room in St. Joseph's Hospital. Her condition was as follows: Weight probably sixty pounds;

temperature at 4 P.M. 102½°; slight œdema of feet; considerable ascites; marked tympanites and slight pitting of skin over the epigastrium.

The diagnosis was tuberculous peritonitis, with probable ulcer in the bowels. The following morning operation was performed. The incision was made midway between ensiform and umbilicus, and had nearly severed the fasciæ when the omentum appeared, and I supposed I had rather quickly cut through the exceedingly thin abdominal walls into the peritoneal cavity; but on exploring a tense ring was found, from which the omentum emerged and spread out in the sub-peritoneal space a circumscribed distance, with a diameter of nearly two inches. It was adherent to the under surface of muscular layer and to the adjacent layer of peritoneum. Forming part of the hernial mass was the ligamentum teres. The adhesions were freed, the ring nicked, and, deeming it best, the omentum with ligament amputated. The abdomen was then quickly explored. The peritoneal and retro-peritoneal glands were enlarged. A mass in the right region was brought to the opening and found to be the cæcum and small gut with a large adjacent glandular mass. I determined an ulcerating tubercular condition located in and about the cæcum, but dropped it back and closed the abdomen after inserting a drainage-tube above the symphysis. Thirty-five minutes only were consumed, but the patient was almost pulseless, and collapsed. The operation was begun at 10 A.M., with a temperature of 97°, and the temperature continued low until 2 P.M., when the patient began to react. The patient did very well until the third night, when an exhausting, profuse diarrhœa occurred. She died the fourth day after operation.

A limited post-mortem was made an hour after death. The incision had healed perfectly. The mesentery was one large nodular mass. The cæcum, as I show you, is ulcerated on its inner surfaces completely for a distance of one and a half inches from the ileo-cæcal valve, which is also ulcerated and contracted, the ulceration extending into the small bowel half an inch. All the peritoneal glands are nodular.

This case earlier in its history would probably have been a curable one. She was a moribund case at time of operation, and was so considered, yet it was judged feasible, with the disease located in the abdomen and the lungs almost free from disease, to give her a chance, which she accepted. She was too low to react.

The etiology is obscure. The spell of sickness she had in June, 1892, was considered typhoid by those attending. I submit the specimens to see if any of the members can find anything indicative in the ulceration of a primary typhoid ulcer. It would be interesting to know if the constant contusing of the abdomen was a factor in the tubercular process

that developed. I consider the occurrence of sub-peritoneal hernia of omentum and ligamentum teres a rarity. *January* 12, 1893.

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30. *Two cases of membranous enteritis.*

Read by Dr. S. Solis-Cohen.

The essential clinical feature of the condition best known as croupous enteritis, or membranous enteritis, is the discharge from the bowel of mucoid, fibro-mucoid or fibrinous casts; the essential pathological feature being, of course, the formation of these casts.

The affection is not common, yet, as Da Costa,[1] in his classical study long ago pointed out, it is hardly one to which the term rare may strictly be applied. I have encountered some half-dozen cases in the course of ten years. Two of these were in young hysterical females, not my patients, whom I saw on a few occasions, in the absence from the city of the attending physicians, and at the request of the latter. One patient was a middle-aged and hysterical woman, who presented herself at the medical clinic of Jefferson Medical College Hospital, during my service as chief assistant; the fourth case was likewise that of a middle-aged and hysterical woman, who applied for treatment at my dispensary service in the Philadelphia Polyclinic. (The early records of this clinic were unfortunately lost or mislaid in moving to the new hospital building, and I have no note of the case except in my index of diagnoses.)

Specimens of the discharge from the two other cases are presented this evening. Both patients are males and the cases present sufficient interest to be placed on record.

Case I. *Membranous enteritis simulating perityphlitis.* D. W., 33 years old, a blacksmith, was sent to my colleague, Prof. J. M. Baldy, at the Philadelphia Polyclinic, by G. A. Beck, of Danville, Pa., with a request to place him under the care of a medical clinician; and by Dr. Baldy referred to the medical ward, then under my care, February 29, 1892.

From the letters of Dr. Beck and the notes taken by Drs. Gibbon and Knipe, resident physicians, the following account of the case is condensed:

About three years ago the patient began to suffer with severe pain in the hypogastric and right iliac regions. The pain occurred in paroxysms,

[1] Amer. Journ. of the Med. Sciences, October, 1871, p. 325.

lasting at first an hour or two; afterward, with slight intermissions, for several days; the intervals between the attacks varying from about a week to so long as two months; the severity progressively increasing.

No information could be obtained as to the character of the discharges prior to August, 1891. At that time a paroxysm of such severity occurred that it was necessary to administer hypodermatic injections of morphine; and the patient was confined to bed for six weeks. The location of the pain, and a tumefaction observed in the right iliac region, aroused the suspicion of perityphlitis, which, however, the subsequent course of events did not verify.

There was passed by stool "something resembling the trachea of a chicken, followed in a few days by something much resembling a piece of intestine; each was about three inches in length. There was no fever and but little gastric disturbance; a tendency to constipation was noted." For some time afterward membranous shreds and casts continued to pass, the discharge being preceded by constipation and paroxysms of colic. The stools did not contain blood.

After this attack, dull pain in the iliac region was constant, but not so severe as to prevent sleep. Paroxysms recurred at intervals of from one to six weeks, and lasted with remissions for one or two days. At their height morphine had to be given. With the passage of membrane the pain moderated, and with its disappearance, the pain ceased. The patient continued in robust health; his family and personal history were devoid of pathological significance.

On admission the patient was a robust, well-nourished man, presenting nothing abnormal to physical exploration. He complained of a dull pain in the right iliac region, which was not sensitive to pressure. He had not had a stool for a few days. An enema of turpentine (f℥j) and magnesium sulphate (℥j) in warm water and soapsuds (Oij) was ordered to be given nightly. The hardened feces, and later the unformed fecal material, thus brought away presented nothing noteworthy.

On March 2, 1892, the patient complained of an increase of pain in the right iliac fossa, which was now sensitive to pressure; and a peculiarly sensitive spot was found in the location known as McBurney's point. There was impairment of resonance over a somewhat irregularly pear-shaped area in the situation of the cæcum, and to palpation a sense of doughy induration was given.

On March 4th, at 5 A.M., the patient was awakened by griping pain beginning to the right of the umbilicus and extending into the right iliac region. Somewhat relieved by hot stupes, it persisted until 7 A.M., when, with much straining, a stool was passed, consisting of unformed

feces and fragments of mucous and membranous casts. The enemata was continued, but brought away no more membrane, only unformed feces and strings of mucus. Slight tumefaction in the right iliac fossa persisted.

The patient was free from pain for six days, when, at 10 P.M., pain, less severe than on the previous occasion, began, as before, at the umbilicus and radiated into the right groin, causing retraction of the right half of the scrotum. The tumefaction was observed to increase, and the abdominal muscles became tense. In about three hours the paroxysm terminated, with the discharge of a small quantity of softened feces with much more mucus and membrane than in the stool of a week earlier. There was much straining at stool. With the passage of the membrane, abdominal tension relaxed, pain ceased, and the tumefaction became much less marked.

Three days later intermitting attacks of sharp shooting pain occurred for about an hour in the forenoon, and recurred at 3 P.M., lasting for about half an hour, when membrane was passed in a few fragments aggregating two inches by two inches, and the pain ceased. On the following day nothing abnormal could be found on abdominal exploration.

Four days later the membrane presented was passed, without straining, after the enema. It is flat, thick, tough, white; toughness and whiteness having become more marked since placing it for preservation in diluted alcohol. The patient complained of dull pain in the hypogastrium, and of slight vertigo. From this time forward no unpleasant symptoms occurred and no membrane was passed. As the patient could not be assured that no attack would occur in the future, he was about two weeks later discharged at his own request.

Drs. Coplin and Bevan examined the membrane passed March 4th and March 10th. They report that "the casts are made up almost entirely of fibrinous exudate in which are found large numbers of leukocytes and epithelial cells. From the mucous surface at some points will be found evidence of the sloughing of the mucous membrane. There are also evidences of young bloodvessels in the membranous casts. In some points hemorrhage has taken place."

These appearances seem to point to the fact that the process is, in part, inflammatory, leading to the formation of a pseudo membrane, which becomes partially organized and then separates by superficial necrosis. Whether the tumefaction in the right iliac region was due to obstruction of the cæcum or colon by accumulation of the products of desquamation, or whether the seat of the morbid process was thus indi-

cated, I do not pretend to say. The size of the casts, however, would indicate their formation in the small intestine.

Case II. *Pseudo-membranous enteritis in a lithœmic patient presenting symptoms of vasomotor ataxia.* X. Y. Z., an attorney, about 32 years old, has suffered for some two years, following a great grief, with symptoms termed by various American and European clinicians of eminence as those of "gastric catarrh," and of "neurasthenia." There are in his case some important features not relevant to the present communication, except that they indicate a highly neurotic constitution. When I saw him some four months ago, he was much emaciated, obstinately constipated, passing small quantities of urine of high specific gravity, containing uric acid, urates, phosphates, and oxalates in excess, red blood-cells, no albumin, no sugar. This oliguria was doubtless due to the dry diet advised by a former attendant, as it soon disappeared when a normal quantity of water was taken; though the uric acid, urates, oxalates, and hæmocytes did not disappear until appropriate treatment had been continued for some time.

The man's sickness began with frequent attacks of vertigo, which had incapacitated him for the duties of his profession; his circulation was poor, so that hands and feet were almost constantly cold; and just before one of his "spells" the lips and nails became blue, the skin of hands and face pallid. I was summoned while he was suffering with a paroxysm of abdominal distress which he said could not be called "pain" or "colic," though he knew no other term by which to describe it. It had ceased when I first saw him, about two hours later.

I cannot say what excited the suspicion of membranous enteritis; probably it was the hypochondriasis, the constipation, and evident intestinal spasm. At all events, failing to elicit a history of the passage of casts, the patient was asked to observe his stools; nightly enemata of warm water with soap, salt, and turpentine, being prescribed together with other treatment.

For some two weeks nothing was found in the stools except ordinary mucus; then on one occasion membranous shreds were discovered after an enema, but were not saved; some days later, a mucous cast, fully eighteen inches in length and about three-eighths of an inch in diameter, was passed; the patient being under the impression that it was a worm, which, indeed, to the untrained eye it resembled. It was composed of semi-translucent, whitish, gelatinous material, manipulable with difficulty, showing no structure under the microscope, except that it contained a few small round cells, lymphoid in appearance; it dissolved readily in alkalies. I regret that its coagulability with acid was not tested.

About a week or ten days later the membrane exhibited was passed. Similar discharges, gradually lessening in quantity and frequency, continued to be observed at intervals for about three weeks. There has been none discovered for more than five weeks past.

Unlike all other cases that I have observed, in this instance these matters were found in ordinary, well-formed stools, that were passed without straining and spontaneously; the discharges were not preceded by colicky attacks or pain of any kind; for the patient's general condition is steadily improving under treatment, so that he is not constipated, is much less hypochondriacal, not at all neurasthenic, eats well, sleeps well, attends to his office and court practice, and has had but two vertiginous spells since coming under my observation.

In this case I think we have a much earlier stage of the affection than any that I have previously encountered, or of which I have read the report. The diagnosis made as to the patient's general condition is lithæmia, vasomotor ataxia, gastro-intestinal catarrh, with marked intestinal indigestion. The membranous enteritis I am inclined to look upon as a sequel to the intestinal catarrh, conditioned by the disturbed state of the trophic and vascular system.

The fact that hysteria or hypochondriasis is almost invariably associated with the condition, lends color to the theory that it is literally a trophopathy of neurotic origin, and its most probable mechanism is disturbed vascularization. Even in the other case I have reported in which inflammation probably exists, the paroxysmal occurrence of the attacks indicates a neurotic element.

Bacteriological investigation was instituted by Dr. Coplin, but his results were not decisive, probably owing to the contaminated state in which the discharges reached him. I am not inclined, however, to regard the condition as in any particular due to microbes. Ordinary intestinal irritation or catarrhal inflammation is its probable starting-point, a neurotic constitution its determining factor.

Dr. Eshner: I have had an opportunity of examining the specimens of membrane of which Dr. Cohen has spoken, and by a strange coincidence I have also received another specimen of the same kind from another gentleman. To the naked eye the specimens appear as yellowish-white shreds of membrane of various sizes and of rather gelatinous, translucent aspect. They are fragile and rather difficult of manipulation, tearing and breaking readily. Treatment with sodic hydrate causes an increase in translucency, while treatment with acetic acid causes them to shrivel. Under both conditions they undergo a diminution in size. Histological sections stain very irregularly, and present no

organized structure. Here and there numerous round cells can be seen irregularly distributed upon a finely granular and rather homogeneous background. The appearances are suggestive of a fibrinous exudation.

January 26, 1893.

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31. *Meckel's diverticulum.*

Exhibited by Dr. C. A. Hamann.

This specimen was removed from the body of a male fœtus, which was stillborn at about the eighth month. The diverticulum arises from the ileum, at a right angle from the side opposite to the attachment of the mesentery, at a point fourteen inches from the ileo-cæcal valve, the entire small intestine being ninety inches long. It is a little over one inch in length, rather club-shaped (*i. e.*, the base is at the opposite end from the intestinal attachment). The calibre is about the same as that of the ileum, and at its extremity there is a fibrous nodule looking as though a band had been attached to it at one time; these nodules or protuberances, which are not infrequently seen on the end of the diverticulum, are, according to Hening, the remains of the vitelline vessels. There is a mesentery running almost to the tip of the diverticulum.

It may be proper to mention that, according to Ahlfeld and others, persistence of the vitelline duct is a causative factor in the production of some of the various forms of atresia ani; the traction which is exerted by the duct, or rather the resistance it offers to the proper development and descent of the hind-gut, causes the latter to fail to communicate with the epiblastic ingrowth which forms the anus.

February 9, 1893.

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32. *Intestinal obstruction due to a gall-stone.*

Exhibited by Dr. Alfred Stengel.

By the kindness of Dr. Wm. Pepper, I am enabled to present this unusual gall-stone, which was sent by Dr. Guthrie, of Wilkes-Barre, with the following history:

"Mrs. O., aged 50 years, was a stout woman, of good family history, and herself in easy circumstances. When first seen she was in bed; there was no fever and no pain or tenderness over the abdomen. There

was, in fact, no definite symptom, excepting persistent vomiting, especially after ingestion of food. The suggestion that gall-stone might be the cause of her symptoms was ruled out by the absence of any history of biliary trouble. I did not see her after my visit of December 12, 1889. She gradually became exhausted and died a week later.

"At the *autopsy* a large gall-stone was found in the ileum, about eighteen inches from the ileo-cæcal valve, with complete occlusion of the gut. Pressing it forward toward the valve it was caught and could not be forced onward, but it could be pushed backward toward the pylorus to any extent. The gall-bladder was found adherent to the duodenum, and upon separating the adhesions there was found an opening from the gall-bladder into the duodenum, through which the stone had passed."

The stone, as you see, is about an inch and three-quarters in length, of elliptoid shape, and about an inch in its smallest diameter. The outer covering is light-brown, but the surface of section, seen where it has been divided, is lustrous and white, and presents the beautiful radiating appearance commonly seen in cholesterin stones. The weight of the stone is very slight, as we generally find in these cases. Clinically, the entire absence of pain, jaundice, or other hepatic symptoms is very important.

Dr. Eshner also showed several gall-stones. One was obtained from a case of protracted jaundice with hepatic intermittent fever, in which the common duct was completely occluded at the junction of the hepatic and cystic ducts. Other specimens were obtained from various cases of jaundice. *February* 23, 1893.

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33. *Cystic disease of the liver.*

Exhibited by Dr. Wharton Sinkler.

The specimen I exhibit was removed from a woman aged 35 years. Five years ago the woman became ill, with violent pains in the right hypochondrium, without vomiting or jaundice. Two years later a similar attack occurred, and another one year ago. A very severe attack occurred three months ago, and from this the patient never wholly recovered. Six weeks ago she noticed that the base of the thorax was enlarging, and on measurement an increase of girth of three or four inches was determined. Ten days ago I found decided bulging of the base of the left chest, and a distinct tumor, but no hydatid fremitus could be detected. There was dulness up to the fourth rib and two or

three inches below the costal margin. It was decided to open the abdomen, and the operation was performed by Dr. W. W. Keen. During the etherization the pafient vomited considerably, and it was noted that the tumor had changed in shape and decreased in size. A rupture was therefore suspected. As soon as the abdomen was opened a rent was detected in the cyst wall and a daughter cyst was detected within. The fluid of the cyst was somewhat turbid, and on microscopical examination showed crenated red corpuscles and certain amœboid bodies. There was also found a large number of bodies which remotely resembled hooklets, but without the shorter projection, and which became dissolved or otherwise disappeared from view in the fluid on subsequent examination. They were evidently not echinococcus hooklets. The patient died shortly after the operation. The liver presents a ruptured cyst from which several quarts of fluid had escaped, and around this a number of smaller cysts projecting outward, not inward, as is frequently the case with hydatids. The lining wall of the cysts is smooth and tough.

I am at a loss to explain the nature of these cysts. I had suspected echinococcus, but the appearances here are certainly not what we are accustomed to see. *March* 23, 1893.

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34. *Irreducible inguinal hernia, containing the ileum, ileo-cœal valve, caput coli, and vermiform appendix.*

Exhibited by Dr. Henry W. Cattell.

The specimen was situated on the right side in a male aged 62 years. The sac contained the commencement of the colon, the vermiform appendix, the ileo-cæcal valve, and a small portion of the ileum. The vermiform appendix came off posteriorly, pointed toward the symphysis pubis, and was patulous throughout. There was also a right-sided hydrocele. In my opinion, on account of the anatomical relations of the parts, the growth must have descended rather slowly.

I am indebted to Drs. Davis and Augé, resident physicians at Blockley, for assistance in the performance of the autopsy.

March 23, 1893.

35. *Gall-stones.*

Exhibited by Dr. J. Hendrie Lloyd.

M. A. M., a white woman, aged 74 years, was admitted to the Philadelphia Hospital February 9, 1893. She was a feeble old woman, very much emaciated, with left-sided hemiplegia and marked motor aphasia. The left arm and leg were contractured. The hemiplegia was of a year's standing. She had been in bed a long while, was very despondent, and did not eat. No change took place in her condition until March 20, 1893, when at the evening visit she was found stupid and incoherent.

The following night she fell into a stupor, from which she could not be roused. Her temperature, normal in the beginning, soon rose, reaching $103\frac{2}{5}°$. Cardiac stimulants and nutrient enemata were given. The coma deepened; there was twitching of the muscles, but no new paralysis. Death took place on the 25th, four days after the development of coma.

The autopsy revealed a sanguineous cyst in the left side of the brain, explaining the paralysis of the right half of the body. On the right side there were recent subcortical and ganglionic hemorrhages. The arteries were very atheromatous.

The gall-bladder contained a large gall-stone, which was smooth and impacted, but there was no occlusion of the duct, and bile flowed on pressure. There were a few small light-colored stones also. The stone weighed 7 grs. It was interesting to find that there had been absolutely no history of hepatic pain or any manifestations of stone in the ducts.

April 13, 1893.

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36. *Hydatid cyst of the liver with secondary endocarditis.*

Exhibited by Dr. W. E. Hughes.

B. P., aged 46 years, had an illness at twenty-seven, during which his entire body was swollen. Had malaria at thirty-four, lasting four months; has also had rheumatism. Two weeks before admission to hospital he had a chill, followed by acute pain in the right hypochondriac region radiating to both shoulders. Heart area increased. At aortic cartilage an exocardial sound heard, also a systolic murmur.

Urine normal at first, but later contained albumin, with no casts. Bowels constipated, stools light yellow. Pain on pressure in the epigastric region. Liver enlarged, hepatic dulness extending from the fifth rib in the mid-axillary line to two inches below the umbilicus. Nodule felt on liver in axillary line. Nose of spleen felt below the ribs. About three weeks prior to death the feet became œdematous, the œdema extending up the leg, thigh, and trunk as high as the base of the chest. Shortly after this purpuric spots appeared on the external aspect of both thighs. Patient died suddenly of heart failure.

Post-mortem. Both pleural sacs obliterated by old adhesions. Pericardium greatly thickened and both layers glued together, although the adhesions were readily broken down. Heart weighed thirty-seven and a half ounces. Walls relaxed and cavities filled with blood. Stenosis of aortic orifice, one valve covered with vegetations on the cardiac side.

Kidneys swollen and capsules adherent.

Liver. Weight, five pounds ten ounces; swollen and granular. Cyst found on upper surface of right lobe, about an inch and a half in diameter, and containing a semi-purulent liquid, but presenting the typical membranous walls of a hydatid cyst.

Pathological diagnosis. Dilatation of heart; stenosis of aortic orifice; contraction of kidneys; hydatid cyst of liver; and pericarditis.

May 11, 1893.

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37. *Cancer of œsophagus.*

Exhibited by Dr. Henry W. Cattell.

The patient, a male, aged 57 years, from whom the specimen was obtained, died in the ward of the University Hospital under the care of Dr. Pepper. The patient had been admitted to the ward some time previously, and had complained of inability to swallow solid food. This had been preceded by a period during which he had aversion from food but not inability to swallow. There was emaciation, and toward the end of his life marked cachexia, and a stricture was discovered by the sound. The diagnosis of cancerous new-growth was therefore easily made in the absence of syphilitic history or other indications.

Autopsy. The body was greatly emaciated. Post-mortem decomposition had already set in and the abdomen was scaphoid. The notes on the cancer alone are quoted.

Œsophagus. A flat, longitudinal, tabular growth is found in the œsophagus a little beneath the cricoid cartilage, having the dimensions of an inch in length by three-quarters of an inch in breadth. It is irregular, raised several lines above the surface, and extends into the surrounding tissue; the nodules being small and well circumscribed. The glands situated in this region are also involved. One and a half inches below this is a second, longitudinal mass, divided by the incision into two parts, the one on the right side being three and a half inches long by one inch in breadth, the portion on the left being longer, slightly less broad, and ending in a fungoid mass, which is interruptedly continued by an irregular path of new material to the stomach. The peribronchial glands are enlarged, hard, and in spots infiltrated with the new-growth. The bronchi are infiltrated from without, but show no changes from within.

On the anterior surface of the stomach at the lesser curvature, near the cardiac end is a large nodular mass, nearly circular, about two inches in diameter. The growth has infiltrated through all the walls of the stomach, and the lymphatic glands both here and for a considerable distance downward. The centres of several of the glands show caseation. Posteriorly the pancreas, near its centre, is slightly attached to the new growth and is somewhat infiltrated. The liver and vessels, and the pyloric end of the stomach are not affected. There are some other scattered circular nodules in the region of the cardiac end of the stomach. The lumen of the œsophagus has never been entirely closed, but the taking of food must have been rendered extremely difficult by the largest mass in the œsophagus and the one at the cardiac end of the stomach. A microscopic section shows the growth to be cancer.

May 11, 1893.

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38. *Cancer of the gall-bladder.*

Exhibited by Dr. E. L. Vansant.

Woman, aged 72 years. Previous history good. General debility; but fairly well nourished. No cachexia or jaundice. Heart sound; pleuritis of left side; old bronchitis. Liver was enlarged and extended two or three inches below the ribs. A hard nodular mass was found about in the median line, which moved with the respiratory movement. No vomiting of blood. Some œdema of feet. Urine contained albumin and casts.

Post-mortem. Large nodule found on liver and small ones scattered over it. Gall-bladder much involved. Stomach entirely free. Pancreas free, some slight involvement of mesenteric glands. The growth evidently started in the gall-bladder, and hence the nodules of the liver were secondary. Kidneys showed chronic Bright's disease; cortex small.

There were a few gall-stones in the gall-bladder, and the interesting question comes up: are the stones the cause of cancer by the irritation they occasion, or the result of obstruction to the flow of the bile due to the cancerous disease of the gall-bladder? For my own part I regard the stones as the probable cause, though it is very difficult to prove this position. *May* 11, 1893.

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39. *Cancer of the stomach with involvement of the adjacent organs and large sub-peritoneal cyst.*

Exhibited by Dr. Henry W. Cattell.

H. G., aged forty-three years, was found by the attending physicians, Drs. Parke and Mitcheson, to be suffering with continuous pain in the right hypochondriac region, also much vomiting, especially after eating. He had been sick for some time and had been under treatment at two hospitals, but without benefit. He had previous to the attack been a strong man, but a hard drinker, which gave him, as he said, occasional attacks of dyspepsia. His occupation was that of a huckster, and he was out early in the morning before meals, and for many years was in the habit of drinking upon an empty stomach. In addition to the pain and nausea, he was suffering from great dyspepsia and ascites, which had been accumulating for about two weeks. He was promptly tapped, and about four quarts of a dark colored liquid (like lager beer) was withdrawn. The patient was greatly relieved next day, but very weak; said he had been tapped upon two previous occasions at the hospitals. The patient was greatly emaciated, and pain and nausea continued, the matters vomited being of a dark yellowish color, and extremely offensive. At no time during his illness did he vomit blood, or anything that would suggest blood. Upon percussion the liver area seemed diminished, but an accurate definition was difficult, owing to the dropsical effusion. Auscultation did not reveal any heart lesion. Family history good. Throughout, the treatment was morphia, to relieve pain, and bismuth subnitrate, and carbolic acid, to allay the nausea. A mercurial purge was now and then ordered, to relieve the bowels.

Shortly after the last tapping, the fluid again commenced to accumulate, but as the patient was extremely weak tapping was deferred. The pain over the region of the liver continued, and at times was so severe that one-quarter grain doses of morphia every two hours were required to relieve it. Small doses of stimulants were occasionally administered to strengthen the patient, who was failing very rapidly. During this time the patient suffered most distressing nausea, and no food whatever would remain on his stomach. Patient was greatly emaciated and continued to grow weaker, and finally died of exhaustion.

At the post-mortem cancer of the stomach, with involvement of the adjacent organs, was found. A large sub-peritoneal cyst was found below the stomach the size of a child's head, and containing a clear fluid. *May* 11, 1893.

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40. *Carcinoma of the stomach and liver.*

Exhibited by Dr. D. J. M. Miller.

The patient from whom the specimens were removed was a man of advanced age. He had suffered with pain after eating, and was cachectic. The liver was somewhat enlarged. He had vomited frequently, but the matters ejected contained blood on but one occasion.

At *autopsy* the stomach was found adherent to the tissues posteriorly, and was much contracted. The anterior wall was about a quarter of an inch thick; it was tough and more or less fibrous. The pyloric end and nearly two-thirds of the whole organ were densely infiltrated with cancerous growth. Here and there on the posterior wall there were spots of ulceration and hemorrhagic infiltration, and there had been some hemorrhage into the tissues back of the stomach. The liver contained small spots of whitish color—doubtless beginning cancerous growths; but there were no large nodules. *May* 25, 1893.

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41. *Volvulus due to extra-uterine pregnancy.*

Exhibited by Dr. Henry W. Cattell.

The extra-uterine pregnancy was on the right side, and had probably occurred some seven months previous to the patient's death, as at that time she had had symptoms which might have been attributed to this

condition. About two inches from the ileo-cæcal valve the ileum was firmly bound down to the mass on the right side, though the gut was patulous. About four feet farther up was a band of connective tissue, resembling in character the adhesive material by which the ileum was held down to the mass. This band was also attached to the intestine some seven or eight inches farther up. By means of this thread-like band a portion of the small intestine which was placed beneath and, turning upon itself, had passed over and above, was caught, and the onward passage of the contents of the bowel was hindered. The bowel above this point was red, swollen, and angry looking, though not gangrenous, and contained a considerable amount of semi-liquid material. The point of constriction lay in the median line a little below the umbilicus. In my opinion, the gut had been formerly attached to the sac of the extra-uterine pregnancy in the same manner as the lower portion of the ileum, and becoming loose, it was capable of moving freely inside of the abdominal cavity. The case had been diagnosed by Drs. Stryker and Evans, the attending physicians, as one of intussusception or of volvulus, and the patient had been removed to the hospital in order that she might be operated upon in case of necessity. Unfortunately, death took place almost immediately after her entrance into the hospital, and the band was not loosened by the surgeon's knife and the life of the patient saved. *May* 25, 1893.

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42. *Carcinoma of gall-bladder and pylorus.*

Exhibited by Drs. A. A. Eshner and F. K. Brown.

Mrs. B., aged 63 years, gave a good previous history till one year ago, when she had an attack of gastro-intestinal catarrh associated with marked debility. She never seemed so strong after this attack, though she never complained of any unpleasant symptoms till April 15th. She then complained of extreme nausea, but no vomiting. No pain in abdominal region, except on deep and continued pressure in right hypochondriac region, where she complained of tenderness. No tumor could be felt on careful palpation. Tongue coated in the centre with thick, yellowish deposit; edges and tip red. The tongue cleared up in a week, and afterward remained red, glazed, and at times dry.

The complexion was fair; there was no yellowness of conjunctiva; no cachexia. No headache, and no abnormal thoracic symptoms declared

themselves. The patient was restless and slightly delirious at night. Temperature, repeatedly taken, varied from 99.2° to 99.6° for four days. No chills. Urinalysis failed to reveal any albumin, sugar, or bile pigment. The urine was high-colored. Bowels obstinately constipated. The liver dulness was normal.

The symptoms remained unchanged for four days, when, the bowels being moved by the enema, temperature declined to normal and remained normal until the last week, when it was subnormal for four days, returning to normal a couple of days before death.

All laxatives failed to act, and bowels had to be opened by forced enemas. No vomiting till the end of first week, when she vomited large quantities of partly digested food and mucus, but no blood.

For about two weeks vomiting occurred every second morning; after that for five or six days every morning; and from then on till death, two or three times a day.

During and after the fourth week she had slight and transient jaundice. After the third week the abdomen on palpation appeared boggy, and at times a soft, movable mass could be felt moving from the right hypochondriac to the epigastric region.

The delirium was low and muttering and continued during the whole course of the disease excepting occasionally during the second week, when she would get a whole night's sleep without delirium. There was no œdema of feet or limbs. The heart was regular and the sounds clear and distinct until the last week, when they became feeble and would occasionally intermit.

Autopsy. The body was much reduced and slightly jaundiced. The abdomen was completely flaccid.

The *liver* was displaced downward three fingers' breadth. The gall-bladder was at once visible below the margin of the liver.

The *stomach* was also displaced downward. Both lungs were slightly adherent at the apices and the lobe of the left was collapsed. Right somewhat œdematous; both congested and cyanotic at bases. There was puckering at the apices, but no appreciable deposits.

Pericardium contained three or four ounces of slightly turbid fluid.

Heart dilated. Cavities contained chicken-fat clots. There were calcareous deposits in left auriculo-ventricular ring, the valves being little thickened and competent.

There were a few atheromatous patches in the ascending aorta, and a large atheromatous plate at the junction of the ascending and transverse portions. The stomach was greatly distended and a large portion lay in the left hypochondrium. The ontents were of a greenish color.

The walls were thickened and covered by mucus, and at the pyloric end there was some contraction, though the orifice was still patulous. The duodenum was narrow, scarcely permitting the little finger to pass, and its walls were thickened. The intestine about the ileo-cæcal valve was also slightly indurated.

The *liver* was enlarged, and the gall-bladder, which projected below the edge, was very hard to the touch and contained three faceted stones of moderate size. The ducts were patulous, but the wall of the ducts and bladder were considerably thickened and indurated. The liver on section was found to be friable, with dilated bile-ducts, and here and there little pockets containing bile-stained muco-pus. The pancreas was large and hard; the splenic vessels along its edge were calcified.

The point of special interest in this case was found in the condition of the gall-bladder and pylorus. Is there any evidence of scirrhus? Certainly it must be admitted that the clinical course was not that of carcinoma; but the induration of the pylorus, duodenum, and gall-bladder was highly suggestive. On microscopical examination of sections from these parts the great proliferation of epithelium within the tissues is striking; and, though no definite arrangements in nests can be discerned in the section from the pylorus, the existence of such in the gall-bladder, and the atypical epithelial growth manifest in all make the diagnosis of carcinoma almost certain. Still, a hasty examination of the pyloric section might be urged as the cause of the failure to discover the malignant nature of the process. *June* 8, 1893.

III. THE VASCULAR AND HÆMATOPOIETIC SYSTEMS.

1. *Aneurism of the aorta with rupture.*

Exhibited by Dr. H. F. Formad.

The three specimens which I show are very similar. In each there is a small aneurismal dilatation at the root of the aorta, which has ruptured into the pericardial sac. There is but little atheromatous change in the vessel in any one of the three. Two of the specimens come from workingmen who did considerable heavy lifting; the third is from a prostitute. In all three cases death occurred during sexual intercourse. Death was sudden and the pericardium was full of blood.

Dr. Packard recalled a case at the Pennsylvania Hospital in which death was delayed four hours after rupture.

The President also had seen a case in which death was slow, the patient having walked to the hospital, and lived several hours after the time when the rupture is supposed to have occurred.

October 22, 1891.

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2. *Aneurism of the aorta.*

Exhibited by Dr. J. H. Musser.

Maggie McG., aged 49 years, a seamstress, widow. Family history was negative. She came to America in 1852. Has had nine children and two miscarriages. Patient states that she was well until April, 1891. She was then taken sick suddenly with chills, cough, dyspnœa, pain in the chest, and headache. This was an attack, she says, of influenza. Cough persisted, and was later accompanied by muco-purulent expectoration, with blood once or twice. There was some loss of flesh and cold night-sweats. Patient suffered most from dyspnœa, which prevented her lying down, and from pain in the chest. On the right side percussion was dull along the margin of the sternum as far out as the junction of the ribs with the costal cartilages. Breathing exaggerated in inspiration, harsh, and prolonged. The expiratory sound was wheezing, suggesting asthma or obstruction to a bronchus. Posteriorly, bronchial râles and

wheezing sounds were heard. Over the upper and second part of the sternum percussion was dull, and also from second rib to clavicle on the left side. Inspiration very feeble, almost absent. Expiration feeble and prolonged. Coarse bronchial râles heard quite generally over the left chest. Percussion resonance good, with the exception noted above. Great dyspnœa, and inability to phonate became marked. Heart-sounds, free from murmur ; second, slightly accentuated. On full expiration a slight pulsation of the entire chest-wall was made out.

Autopsy. Body of good size, very anæmic woman; no œdema; scar in left groin; staining from an old eruption on tibiæ, but no scars.

Lungs. Left—no cavity or phthisical process, but a muco-purulent bronchitis. Right—also the seat of a bronchitis, as shown by the muco-purulent contents.

Heart and vessels. Inferior vena cava much dilated; auricles well distended with blood; slight thickening of the posterior mitral segment. Tricuspid valves in good condition; aortic valves slightly thickened. Coronary arteries coming off normally, but slightly atheromatous. Aorta showed a few calcareous plates.

An aneurism was discovered in the region of the transverse part of the arch of the aorta, extending from an inch above the valves to three-quarters of an inch beyond the left subclavian vessel. The lower part of the sac was filled with dense, laminated clots, so that the blood passed through the upper part. The aneurism compressed the recurrent laryngeal and pneumogastric nerves of the left side, the œsophagus, and trachea, but especially the left bronchus. *October* 22, 1891.

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3. *Abscess of the heart.*

Exhibited by Dr. H. F. Formad.

The heart which I exhibit was taken from a man who died suddenly while dancing. There is present a well-marked purulent myocarditis—an abscess, the size of a large bean, being seen in the left ventricular wall, posteriorly. The contents of this were sero-purulent and somewhat bloody. There were many ecchymoses in the pericardium. The valves show slight atheromatous thickening, also the coronary arteries; but the muscular substance, except in the region indicated, is healthy. The weight of the heart was ten ounces. *November* 12, 1891.

4. *Hypertrophy of the heart.*

Exhibited by Dr. H. F. Formad.

This greatly enlarged heart was found at autopsy on an old man, said to have been 99 years old. There is well-marked atheroma of the aorta, but the valves are not diseased. Both sides of the heart were filled with enormous clots. The organ weighed thirty ounces.

November 12, 1891.

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5. *A case of sacculated aneurism of the aorta treated by electrolysis through introduced wire.*

Exhibited by Dr. D. D. Stewart.

The history of my case, involving a number of points of interest, is related somewhat in detail.

F. D. F., American, white male, of spare build, height about five feet seven inches; best weight about 140 pounds; occupation, insurance agent. First seen in July, 1888, then aged 30 years. About two years prior to this date pain had developed in the back in consequence of attempting to carry a heavy load. Lumbar pain thus induced, though slight at first, became severe after three or four days, and so continued steadily for about a month. Subsequently pain was chiefly localized in the left lumbar region anteriorly and posteriorly. Besides a dull, more or less continuous ache, there were lancinating pains in the area of distribution of the upper lumbar nerves. Several months before he was first seen, at a time when the pains were very slight or were absent, he indulged in violent wrestling, an exercise of which he was fond. Immediately afterward, while seated, he felt something give way in the former painful area. Shortly subsequent to this a small pulsating tumor appeared in the upper part of the left lumbar region. There was no history of lues. He was a trifle given to excessive venery, but was otherwise temperate. The bowels were much constipated. Examination revealed a small, expansile, pulsating swelling to the left of the upper lumbar vertebræ. Over it the second sound of the heart was plainly distinguishable, but no murmur. The latter was present somewhat above, at the border of the twelfth rib. There was no pulsation or murmur to be heard anteriorly. The heart was overacting, but otherwise normal. No difference could be detected in the femoral

pulses. Aneurism of the abdominal aorta was diagnosticated, and the patient received into the wards of the Jefferson Hospital. He was put at rest in recumbency, on a spare diet, after the method of Tufnell. Potassium iodide was prescribed at first in doses of gr. x, three times daily, and subsequently in larger amount. The bowels were moved daily by laxatives, and codeine ordered in moderate doses for the relief of pain. The patient could be persuaded to keep at rest only with difficulty, and after a three months' sojourn in the ward, during which the pain much ameliorated and for a time disappeared, though no marked alteration occurred in the aneurism, he left the hospital. As it was thought that rest in recumbency rather than potassium iodide had benefited him, barium chloride was prescribed three weeks before his departure, at first in doses of one-tenth of a grain, and after a few days one-sixth of a grain, with seemingly remarkable benefit in the symptoms. Under this drug (other conditions as before), pain—which, despite every care taken to improve his condition, began to be as severe as formerly—now almost disappeared, and the area of aneurismal impulse somewhat diminished. He could not be persuaded to continue longer in the hospital, as his means were limited, and he felt sufficiently improved to continue his work. He was not seen again until seven months later (May, 1889). He then stated there had been little or no constant pain in the interval, and that he had been able to continue his work without interruption. During this period he had taken the barium chloride in doses of from one-sixth to one-third of a grain. The tumor had not increased in size. In the erect posture the impulse was slight, detectable only by palpation; in recumbency, prone, it was perceptible to the eye, but not nearly so extended as at first, and the sounds were more distant, indicating that partial solidification had occurred. The heart was somewhat irritable, with a relatively accentuated apical second sound. The pulse was: standing, 100; sitting, 96. The bowels, as before, were unmoved without purgatives. Barium chloride was now increased to one-half grain doses.

I subsequently saw him but four times up to October 26th: once in June and three times in October. During this period, contrary to my advice, he actively pursued his vocation, which necessitated a great deal of walking. When seen in the autumn the improvement had ceased to be maintained. Without my knowledge or advice he had increased the dose of barium chloride to three-fourths of a grain a month before I saw him, on October 17th. The area of bulging and impulse was then larger, and the heart was very irritable. Barium was now discontinued. He consented to rest for a few weeks, and pursue the Tufnell

treatment; but, after a short time spent in bed, he returned to work, and was not seen again until September last. He then had been confined to the house for two months, being too much prostrated and the pain too severe to go about. During the interval of two years in which

A. Site and direction of entrance of canula through which wire was introduced.
B. Site and direction of entrance of second needle.

I had not seen him he had taken little or no care of himself. His physical condition had grown progressively worse. Despite this, he continued his employment quite steadily until within a few months of September. More or less gnawing pain was felt in the region of the aneurism, with paroxysmal attacks of left-sided abdominal pain. The bulging posteriorly had reached a length of five and a half inches; its

greatest breadth four and a half inches. There were three large nodules upon it, far more prominent than are shown in the photograph. Bulging began opposite the spinous process of the ninth dorsal vertebra, and extended to the lower lumbar spines. The abdomen and ribs were more prominent on the left side, which measured from mid-spine to mid-sternum one and three-fourths inches in excess of the right side. A marked impulse was perceptible to the eye and by palpation in all parts of the aneurism, the walls of which seemed thin and but ill-protected by clot. A murmur was heard over the most prominent portions. No sounds were detected laterally or in front over the prominent left abdomen. The area of splenic dulness was considerably increased. The heart was irritable, its sounds high-pitched, the first lacking in muscular element: the impulse was slightly lowered and displaced to the left. There was a markedly accentuated pulmonary second sound, without any sign of an obstructive cardiac affection. The pulse was somewhat irregular and compressible. There were also cough, mucopurulent expectoration, and some dyspnœa. Mucous râles were present in the upper part of both lungs, accompanied by diminished resonance on percussion at the left apex. There was an area of anæsthesia extending over the aneurism from about the ninth dorsal spine above and the second lumbar below laterally and anteriorly toward the median line, in a direction slightly downward. Above and below this there was intensely heightened tactile and pain sense, the slightest touch or firm pressure causing much suffering. The superficial reflexes of dorsum, chest, and abdomen on the left side were extraordinarily heightened, very trifling stimulation producing markedly increased response on the same and the opposite side. The right knee-jerk was +, the left + +. Ankle clonus was absent. There was no paraplegia. The pain felt in the back was quite constant, lasting for hours, and was gnawing in character. Excruciatingly severe pain was felt in the left hypochondrium, loin, and abdomen. To relieve this he had been taking codeine in the extraordinary doses of gr. xx to gr. xxx daily. This was ordered discontinued, small doses of denarcotized opium at short intervals replacing it. He was removed to St. Mary's Hospital in September.

During his sojourn there his physical condition improved somewhat, but the area of bulging seemed to grow larger almost daily. As the physical signs indicated rapid advancement, and that rupture of the sac was imminent, the performance of electrolysis through introduced wire was suggested to him as offering a chance of at least slightly delaying the fatal issue, and, perhaps, also promoting euthanasia. He was

told a cure was now impossible. With this understanding he was anxious for the operation. It was done on December 7th last, with the assistance of Dr. Pottberg, to whose mechanical skill I am indebted for many suggestions of value in the technique. Mr. Otto Flemming, who contributed the electrical outfit and needles, was also present. As my prime object in this case—in which a cure could not be expected—was rather to promote prompt formation of firm protecting coagula, in order to retard rupture of the thin-walled sac, than to cure an aneurism of such dimensions, with symptoms indicating advanced implication of vital parts, I chose a rather heavy silver wire;[1] for, on consideration, I concluded a better chance of immediate success lay in the introduction of wire of sufficient calibre to form large, supporting spirals in the sac, thus occupying considerable of its cavity, and affording a framework for clot, than in the use of a thinner, more pliant wire, which, though better calculated to permit of ultimate contraction of organized fibrin and sac walls—apparently not to be hoped for here—might undergo deflection from its course in introduction through impingement on loose coagula already in the sac, thus perhaps rendering it necessary to repeat the process, which from the patient's enfeebled condition would have been impracticable. A canulated steel needle, two and a half inches in length, of sufficient size to permit the passage of the wire, insulated with shellac to within half an inch of its point, was used to pass the wire. The wire and needles were sterilized by boiling in carbolated water. The hot water somewhat softened the insulation, which was subsequently hardened in cool air. I feared to use carbolized glycerin to lubricate the needles before introduction, lest this would also disturb the insulation. The use of unsterilized glycerin or oil was considered unsafe because of the danger of sepsis, so the needles were passed after being simply remoistened in water. The canulated needle carrying the wire was inserted to within half an inch of its hilt. A platinum needle was also used, which was passed until it was thought that the insulated part was fully within the sac. I had primarily intended introducing the canulated needle in the upper, most prominent, and apparently the thinnest part of the sac wall, in a downward direction, as nearly as possible parallel with the long axis of the body, hoping in this way to favor coagulation that would protect the exterior wall, but little guarded by clot. Mr. Flemming had made for me four

[1] "Drawn hard to No. 23 Brown & Sharp gauge." It was so drawn that it would readily form spiral coils. It was afterward kept for several days on a roll two and a half inches in diameter, so that when removed and passed through a canula it assumed separate spirals each of about three to five inches in diameter, a few feet of it covering quite a large area.

platinum needles of good calibre, two inches in length and insulated to the extent of one inch. These I had also intended introducing below and over the most prominent parts of the sac, in a direction perpendicular to that of the canulated one, connected with the same rheophore, thinking that I might thus be able to secure the formation of multiple coagula extending from the wire to the needles and sac wall, and thus nearly consolidate the sac. Unfortunately for my purpose, it was found impossible to insert the canulated needle above in the direction desired, there still remaining some portions of the ribs yet unabsorbed, which interfered with its introduction. A trial was made in two places, but so much pain and disturbance of the sac was caused by my efforts that I desisted, passing the needle below, in an upward direction, with the patient reclining prone.

Two and a half feet of wire were now slowly inserted, the distal end of the wire being first blunted by filing. An effort was now made to introduce the platinum needles. These, however, because of their cutting ends not being spear- or lancet-shaped, could be passed only with the greatest difficulty, the use of oil not being feasible, so that but one of the four was entered. The current was gradually increased through the controller until seventy milliampères were reached, and was maintained at this strength for one hour. The negative (indifferent) electrode was a very large felt plate, which lay upon the right shoulder and scapula, and was kept constantly moist with hot water. Notwithstanding this, and also that its situation was altered as much as space would permit, the large surface covered was the seat of intense pain, so that the current was momentarily opened through the rheostat to place a large folded, thoroughly-wet towel beneath the plate—a method I had before found to be of service when much pain is experienced from the contact of the felt when very strong currents are employed. Pain was unnoticed here after this, and none was experienced at the site of the active pole in either needles or sac. The pulse became quite feeble during the early part of the electrical treatment, but this weakness was not maintained, the patient standing the ordeal well. Considerable difficulty was encountered in withdrawing the needles, due evidently to the firmness of the clot about them. I preferred not to practise a reversal of the current before their withdrawal, as has been recommended in the electrolysis of aneurism, lest the coagulum forming about the anode be softened by cathodic action. By gentle and continuous rotation and slight traction, with lateral pressure on the sac walls, they were removed, being also aided by an ingenious thought of Dr. Pottberg's, that of taking the lid of a stiff pasteboard pillbox, parting it from its periphery to its centre,

and slipping it about the needle's base. Pressure made upon this by the fingers, instead of directly upon the sac, enabled more counter-force to be utilized in withdrawal. Before removing the needles an attempt was made to push the portion of wire yet within the canula into the sac. This procedure does not commend itself to my judgment, as it appears that such a method must inevitably disturb the relation obtained by electrolysis between the wire, clot, and sac wall, loosening the coagulum if attached to the sac. But a small portion had been passed in when blood appeared at the canula's extremity, and at the same time the patient stated that he felt something moving within, the first sensation that had been noticed in the sac. The needle was now cautiously withdrawn in the manner stated, the wire cut close to the skin and gently pushed beneath the latter, after drawing the skin as a cover over the wire.[1] During the introduction of the canulated needle blood spurted on the needle entering the sac wall to about the depth of one-third to one-half of an inch, this indicating the extreme thinness of wall, covered by skin, fascia, and muscle, and supposed to be lined by a certain amount of clot. Hemorrhage ceased on passing the wire into the canula. None occurred on withdrawing the needles. Their site of exit was sealed with cotton soaked in iodoform collodion.

The condition of both needles on withdrawal showed unquestionable clot-formation about them. Both insulated and uninsulated portions of the platinum one, as far as it had penetrated into the sac cavity, were covered with thick, white, tenacious fibrin, in appearance resembling white paint. The steel canulated one was heavily coated with clot, and its uninsulated portion was partly decomposed, a slight touch on the cutting extremity crumbling it beneath the finger.

A few hours subsequently the sac felt warmer than before, indicating inflammatory reaction, and pulsation seemed much less expansile. Considerable pain was felt in the centre and left side of the abdomen, but no more than had been present before the electrolysis. This was relieved by the passage of flatus and feces. Small doses of morphine and chloral were ordered should the pain become unbearable without their administration, the utmost quiet in recumbency was enjoined, and but little fluid in the shape of drink or food permitted. The patient was very uneasy, as he had been before the operation, though for several days pain was not so severe as formerly, but the same amount of opiate was taken. It was impossible to keep him at rest. He tossed about the bed constantly,

[1] The skin had been pushed to one side in introducing the needles, in order to form a valve-like opening.

and could not be restrained at times from walking about the room. Despite this, a remarkable change was noticed in the condition of the aneurism when the latter was examined on the third day. The prominent pulsating portion through which the canulated needle had been passed had sunken to the level of the general aneurismal surface, and transmitted pulsation alone could be detected in it. The whole of the lower part of the sac felt much firmer, and was quite without pulsation, while the extreme upper part seemed to have undergone no change. Yet all parts of the trunk about the sac, above and below, laterally and anteriorly, could now be handled lightly and with varying degrees of pressure without the slightest discomfort on the part of the patient, the heightened tactile- and pain-sensibility having entirely disappeared, as had likewise the much-increased superficial reflexes. The tendon reflexes in the legs were not again tested. For several days the pain seemed less. Little, if any, darting pain occurred, but the gnawing sensation was felt deeply in the abdomen, probably due to erosion of vertebræ, and after the fourth or fifth day it became as severe as before. At the end of the ninth day succeeding the operation, immediately after the patient had been tossing himself about on the bed from the prone to the supine position and had been thumping his left side with his fist—a practice he was addicted to when in pain, and which he insisted upon pursuing, though aware of its danger—he suddenly called for a cuspidor that he might spit, and almost immediately a gush of blood came from his mouth, and in a few seconds he was dead.

A necropsy was made eight hours succeeding death. Body had been placed in dorsal position; it was still warm. Blood oozed from the mouth when turned upon the side. The former aneurismal bulging had disappeared. On opening the abdomen the sac was not discernible without displacing the stomach and intestines. On removal of the sternum the left lung appeared collapsed. The anterior surface of the left pleural cavity was filled with fluid and clotted blood. The right lung was normally distended with air. Pleural adhesions existed on both sides above, laterally, and posteriorly. The pericardium contained a normal amount of fluid. The heart was one and a half times the size of the closed fist. Left ventricle especially hypertrophied. The valves were normal. The ascending and transverse aorta was slightly dilated saccularly, and the seat of extensive atheromatous degeneration. The spleen was six inches in length, five inches in width, and one and a half inches in thickness. Its anterior surface was adherent to the capsule of the left lobe of the liver, two and a half inches of which extended across a good part of the spleen. The anterior and posterior upper margins of

the spleen and about one-half of the upper posterior portion were intimately bound to the aneurism, which lay above it and tended to push the spleen downward and forward.

The sac extended from a point about opposite the right nipple obliquely downward and to the left, across the bodies of the ninth, tenth, and eleventh dorsal vertebræ to the lower edge of the twelfth rib on the left, filling the hypochondrium and a part of the lumbar region. It was twelve inches in its oblique measurement, four inches in transverse diameter above on the right, and six inches below on the left. The aneurism had arisen from the posterior and left lateral wall of the lower thoracic aorta, and subsequently had involved a portion of the abdominal aorta. It had advanced beneath the base of the left lung, and thence downward, the diaphragm forming a partial covering. The anterior wall of the aorta was plainly discernible crossing the sac, the orifice of which measured two inches in length. An effort was made to remove the sac entire without disturbance of its contents, and the left lung with it, into which rupture had occurred. This was found to be impossible without mutilation of the exterior of the body, forbidden by the relatives. The boundaries of the aneurism, including, as they did, vertebræ and the greater portion of the posterior part of the right trunk, prevented the enucleation of the thin-walled sac without coincident removal of these structures. The sac was therefore opened *in situ*. It was yet distended with about a quart of clot and some fluid blood. The site of rupture—a one and a half inch tear—was in the upper left portion, the rip extending into the base of the lung, and thence upward toward the posterior part of the apex of the superior lobe and through the visceral pleura. The wire lay in coils in the sac. The highest portion of the most superficial coil was about two and a half inches below the point of rupture. The distal extremity of the wire lay in the same situation, its point curved somewhat away from the ruptured portion of the sac. The wire was adherent below to the sac wall, but whether by clot or through its proximal portion not having completely entered the sac, I could not discover, as when an effort was made to ascertain this the wire and coagula had become loosened in the cavity through a second attempt being made to remove the sac. Firm clots existed in all portions of the aneurism, with softened ones evidently of very recent origin. The wire was engaged in several large, firm clots which were of so solid a texture that when examined in that part of the sac which was removed they could be separated from sac and wire only with some difficulty.

The bodies of the lower dorsal vertebræ were much eroded, the spinal

cord, protected only by its membranes and thin laminated fibrin, lying exposed in the sac. These vertebræ formed part of the sac wall, as did also the inner surface of the posterior and lateral portions of the inferior ribs on the left. These ribs, in parts, had almost entirely disappeared. All portions of the periphery of the sac contained more or less white, laminated coagulum, some of which had undergone organization. The rent in the sac extended through laminated clot and sac wall into the base of the lung, ploughing for itself a sinus of some size. The calibre of the greater portion of the transverse colon and all its descending portion was much narrowed. The other abdominal viscera were normal. There were cretaceous nodules at the apex of the left lung.

January 14, 1892.

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6. *Leukæmic blood.*

Exhibited by Dr. Alfred Stengel.

I have placed under the microscope a slide showing leukæmic blood stained according to the method of Ehrlich, and in order to explain its features I shall briefly review some of the important points. Ehrlich first pointed out, about 1880, that in the blood of a healthy man there are different kinds of leukocytes, different in their appearances, properties, and origin; and at that time and since he has described methods of staining by which these different forms may be distinguished. According to his investigations there are present in the blood five forms of white corpuscles, distinguishable by their size, the size and shape of the nucleus, the readiness with which the protoplasm and nucleus take on stains, and by the presence of certain kinds of granular matter. To take up the last point first—he recognized several kinds of granules, the more important of which are: (1) coarse, yellowish, highly refractive, oil-like bodies, which are definitely determined by their property of staining intensely with eosin, whence they are called *eosinophilous granulations;* (2) granules which stain intensely with basic aniline stains, such as dahlia or methyl-blue (stains in which the coloring principle resides in the base of the salt, and not, as in the acid stains, in the acid portion), these being called the *basophilic granulations;* and (3) finer granules which are brought out by mixtures of acid with basic stains, such as acid fuchsine with methyl-blue, and which, therefore, receive the name of *neutrophilic granulations.* In normal human blood the eosinophilic and neutrophilic granulations alone are found, the baso-

philic being present only in disease, though occurring normally in the blood of certain animals.

Now the leukocytes themselves, as above stated, are of five kinds:

1. *Lymphocytes.* Small bodies, generally of less diameter than the red corpuscles, having a large nucleus, almost entirely filling the cell and leaving only a narrow rim of protoplasm which contains no granules. The origin of this form is supposed to be the lymph glands. They constitute about 20 or 30 per cent. of all the white corpuscles.

2. *Large mononuclear leukocytes.* Large cells with relatively much protoplasm, also devoid of granules, and a large round nucleus. They are often several times the size of a red corpuscle. Neither the nucleus nor the protoplasm takes stains well. Their origin is supposed to be from the spleen or bone marrow. They constitute, together with the next form, about 6 or 8 per cent. of all the white corpuscles.

3. *Transitional form.* Exactly similar to the last, excepting that the nucleus is somewhat indented and irregular in shape. Their origin is probably the same as the last form. They are considered as a transitional form between the large mononuclear and—

4. *Polynuclear form.* A smaller variety, though still larger than the red corpuscles. The nucleus is smaller but very irregular in shape, being U- or S-shaped, and very frequently broken into pieces. It stains intensely. The protoplasm is densely filled with fine neutrophilic granules. The nucleus is regarded as a long, slender cylinder, which becomes bent in various ways, and often, either as a result of degeneration or our method of preparation, breaks into fragments. Their origin is the spleen, and they constitute from 60 to 75 per cent. of all the leukocytes.

5. *Eosinophilous leukocytes.* Large cells, with large nucleus and the protoplasm filled with coarse eosinophilous granules. They are perhaps derived from the bone-marrow, and constitute about 1 to 3 per cent. of all the white corpuscles.

These are the forms and characters of the leukocytes of normal blood. They vary somewhat in the lower animals, but this does not at present concern us. In leukæmia there is not only the enormous increase of the number of white corpuscles, but there are certain differences in the relative proportion of the different kinds. Thus, as will be seen in the specimen I present, the number of eosinophilous cells becomes particularly conspicuous. Instead of 1 to 3 per cent., there may be 10, 15, or 20 per cent. of all the forms. This is perhaps due to implication of the bone-marrow. In a pure case of splenic leukæmia the greatest increase ought to be in the polynuclear, transitional, and mononuclear forms; in

a pure lymphatic case the lymphocytes ought especially to show increase, whereas the eosinophiles would indicate involvement of bone-marrow. As a matter of fact, I have found the latter abundant in every case of leukæmia which I have examined by this method, and too much importance cannot, therefore, be placed on the determination of the relative proportions.

Now in addition to these normal forms there is a kind of leukocyte in leukæmic blood, not found under other circumstances. It resembles the large mononuclear or transitional forms, but its protoplasm is filled with neutrophilic granules. It has been supposed to take origin from the bone-marrow, and in consequence received the name *myelocyte.* As far as we now know this form does not occur in normal blood. It is often (if not always) present in leukæmia. Another form of abnormal corpuscles sometimes seen in the blood in disease is one in which the protoplasm contains coarse *basophilic* granulations. These cells are very similar to the plasma cells of the connective tissues, or *mastzellen*, as Ehrlich recently termed them; and they are known by the latter name. They occur normally in frog's blood but not in that of man.

The objection has been raised to these methods, that whatever scientific or artistic nicety might be allowed them, they did not furnish us any practical results. This opinion, I hold, is entirely erroneous, and in support of my contention would offer a few instances. A case of leukæmia was reported from Johns Hopkins Hospital by Toulmin and Thayer, in which arsenic, given persistently, caused diminution in the size of the spleen to a remarkable extent and reduction in the number of leukocytes to the normal. Subsequently a relapse occurred, and the case progressed. Now, as Thayer remarks, at the time of reduction of leukocytes physical examination and an ordinary microscopic investigation of the blood would not have established the existence of leukæmia, and yet at this very time the presence of myelocytes and the proportion of eosinophilous cells clearly demonstrated the existence of the disease, though it was then in a latent form. As another example may be cited the infantile pseudo-leukæmia of Jaksch. This disease (if it be an entity) has many of the characters of leukæmia, including leukocytosis, but it differs essentially in its course and nature. This much is known, though we have yet much to learn regarding the entire history of such cases. In a recent communication Baginsky reported several (I believe five) cases, and in none of these were eosinophilous cells conspicuous or myelocytes present, though there was distinct increase in the number of white corpuscles. The differential diagnosis in such a case would rest somewhat, indeed largely, upon the results of the staining methods. These

two instances, I think, are sufficient to indicate that Ehrlich's work has a distinct value in clinical medicine, and I believe that they may lead to further advances in our knowledge.

A few words in regard to the method of preparation and staining may not be amiss. A small drop of blood is pressed between two cover-glasses, which are then separated by sliding one over the other, as is done with sputum and other soft material. The blood is allowed to dry in air and is then heated for an hour or several hours at a temperature of 120° C., by which means the blood is fixed. Another method, and better for practical purposes, I think, is to place the dried cover-glasses in a mixture of absolute alcohol and ether, equal parts, for a half-hour; the specimens are now (either method) ready for staining. The preparation under the microscope was stained with a solution of indulin, aurantia, and eosin in glycerin. The red corpuscles will be seen deeply stained of an orange tint by the aurantia, the nuclei of the white corpuscles and of the nucleated reds are colored dark purplish by the indulin, and the eosin renders the eosinophilous granules a brilliant pink. The staining may be done somewhat more simply by first placing the cover-glass in a saturated solution of eosin in glycerin or in a 1 per cent. solution in 60 per cent. alcohol; then counter-staining with weak hæmatoxylon solution or weak methyl-blue. Numerous combinations have been suggested for demonstrating the neutrophilic and basophilic granules and other constituents, but for practical purposes the two methods given suffice. *January* 14, 1892.

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7. *Abnormal circles of Willis.*

Exhibited by Dr. Joseph Leidy, Jr.

Dr. Leidy presented twenty-five specimens of circles of Willis for the purpose of drawing attention to certain anatomical irregularities which may form an anatomical basis as the explanation of irregular symptoms during the course of cerebral disease.

Of the number presented, there was a marked asymmetrical condition to be noted in each specimen, and anomalous distribution of the vessels in eight of the specimens. Both conditions merit special description. It is to be noted in the first place that there was asymmetry existing in the specimens rather as a rule than as the exception. Though anomalies were seen in one-third of the cases, this condition must be regarded as unusual, a distinction being made between anomalous circles of Willis

and asymmetrical circles—a point which would seem to have received too little consideration heretofore. In a case of multiple thrombosis of the circle of Willis, an interesting specimen was found which illustrated some of these points. The anterior communicating artery was less than a line in length, the anterior cerebral practically having no connecting artery, but opening the one into the other by a perforation at the point of apposition. The posterior communicating upon the left side was fully three lines in diameter, that upon the right being no thicker than a hair and but one-half the length of the other. The middle and posterior cerebral arteries were apparently symmetrical. This specimen was strikingly like one exhibited at a meeting a year ago, a case of double paralysis. The right side first became hemiplegic, followed by a left hemiplegia, the right side recovering the use of the leg and arm. The question was asked whether the rapid disappearance of paralytic symptoms in certain cases might not be due as much to establishment of collateral circulation as to absorption of the thrombus. Such is the rule in other parts of the body, and we know of no anatomical reason why the same might not occur in cerebral embolism and thrombosis.

February 11, 1892.

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8. *Specimens of a case of Hodgkin's disease.*

Exhibited by Dr. F. S. Pearce.

The specimens I show this evening are from a case which occurred in the Presbyterian Hospital, in the services of Drs. O. P. Rex and R. G. Curtin, being reported in the November (1891) number of the *University Medical Magazine.* This is a mass of enlarged bronchial glands, some the size of a walnut, as you see, the whole mass on each side being as large as a lemon. On section, the glands are of uniform consistency. Here, in connection with the trachea, about at its bifurcation and connected with the aorta, is a large mass of glands, the size of a peach, of the same nature as those described. This mass pushed the aorta somewhat forward.

The heart is large, flabby, but still fairly well nourished ; aortic valves atheromatous. The spleen shows no pathological change. The kidneys are of ordinary size and present many light-colored spots distributed through their substance, which are doubtless areas of leukæmic infiltration. The liver is of slightly increased size and presents the same light-colored spots as seen in the kidneys. There were no evident alterations

discoverable in the abdominal glands, in the intestines, or retroperitoneal glands.

In the neck on both sides were many enlarged lymph glands, continuous with the glands of the thorax, extending down under the clavicles and sternum and upward as high as the jawbone. None of the glands were softened, and there was in no place the least trace of a new growth. The disease process was limited apparently to the lymph glands of the neck and thorax (anterior and posterior mediastinum) and groin, except that the liver and kidneys showed evidences of extension of the process.

In the absence of any tubercular deposits, and with the blood changes (anæmia, without increase of white corpuscles in proportion; also, microcytosis and poikilocytosis), the diagnosis of Hodgkin's disease was confirmed.

Dr. Stengel: When we were making the autopsy in the case which Dr. Pearce has detailed I was particularly interested by the relation of the enlarged bronchial glands to the arch of the aorta, and, as you have heard of, the latter was pushed forward. This fact has been utilized by Jaccoud in determining the presence of glandular enlargements of the thorax when the physical signs are obscure. He finds it possible, by deep pressure in the supra-sternal fossa, to feel the transverse portion of the aortic arch, pushed upward and forward by the enlarged glands. This would have been a valuable sign in the present case, and might have led to a complete diagnosis of the conditions present.

February 11, 1892.

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9. *Fatty heart from a case of pernicious anæmia.*

Exhibited by Dr. J. P. Crozer Griffith.

It has frequently been remarked that the heart is extremely fatty in pernicious anæmia, but this change is by no means characteristic, nor is it constant. In this case the fatty condition of the myocardium is very extreme, and I doubt if any of us will recall a more marked instance. The entire muscular substance, as you see, is uniformly yellowish and fatty, though the papillary muscles and the portions immediately beneath the endocardium elsewhere seem particularly affected.

February 25, 1892.

10. *Purpura hæmorrhagica.*

Exhibited by Dr. H. F. Formad.

I wish to relate the history of one case and show a specimen from a second.

Case I. A young woman, aged 20 years, was suddenly taken with high fever in the evening. During the night she began to vomit and became jaundiced, the skin and conjunctivæ being distinctly tinged. Then numerous hemorrhages in the skin occurred. These varied in size from the diameter of a pea to that of a silver dollar. Death occurred by œdema of the glottis. At the autopsy, besides the external hemorrhages, there were seen similar ecchymoses on all of the serous and mucous surfaces. This was true even of the endocardium and the valves. The larynx contained a blood clot.

Case II. A woman, aged 52 years, was an inmate of the out-wards of Blockley. Without premonitory symptoms, she began to have hemorrhages from the mouth and widespread purpura, without fever. Subsequently icterus came on and increased in depth and extent. Death was due to suffocation. At the autopsy much the same condition of things was seen as in the case of the young woman. The icteric staining of the skin and subcutaneous tissues was plainly evident. There were blood-clots in the larynx, with œdema and hemorrhages of the mucous membrane. The purpuric condition also affected the trachea and serous membranes. With the exception of old scars and surface ecchymoses on the liver, no disease of the organs could be made out. Nowhere was anything like a hemorrhagic infarct visible.

These cases are of extreme interest from the standpoint of etiology. Purpura is but a symptomatic expression of various diseases, no doubt; and we must seek to determine the underlying cause. Nervous, rheumatic, septic conditions, and many specific fevers, like scarlatina, variola, etc., are among the causes. The question of a bacterian origin has been of late considered and certain forms discovered, but the whole question is still in much obscurity. *March* 10, 1892.

11. *Aneurism of aorta; rupture.*

Exhibited by Dr. M. Howard Fussell.

The patient from whom this specimen was removed presented himself at the University Medical Dispensary, July 29, 1888. He complained of pain in left chest and back, "rheumatism." On examination, there was prominence of the manubrium. Over this prominence there was dulness and a systolic thrill. There was a diastolic murmur over the prominence.

April 5, 1888, Dr. George Dock saw the patient at home. He had retired feeling well, and awoke about three o'clock with a "salty taste" in his mouth. He immediately coughed up at least one pint of pure, frothy blood. The patient was kept in bed, and there was no recurrence of the bleeding.

He went about his duties as usual after July, 1888. His symptoms were not at all severe from that time until his sudden death, August, 1891. He arose to urinate, and fell back, expiring almost instantly.

Autopsy, twenty-four hours after death. Precordia somewhat prominent, especially near the sternum. On opening the cavity of the chest a considerable amount of serum exuded from the left side. All the organs were displaced to the right. The left lung was floated upward, and when pulled aside, the left thoracic cavity was seen to be filled to two inches above the level of the spine with blood and serum. The liquid when removed consisted of sixty ounces of serum and clotted blood. A mass the size of two fists, occupying the seat of the arch of the aorta, was found to be adherent from mid-sternum in first interspace out on a line to mid-clavicle upward to the middle of clavicle and backward on a level with the first rib. The apex of the right lung was firmly adherent. There were hypostatic congestion and œdema of right lung, and adhesions between upper, middle, and lower lobes. Spleen small, flabby, and pale. Left kidney rather small; capsule slightly adherent. It was mottled, and the cut surface was anæmic. Relation of cortex and pyramids was normal. The mass, which proved to be the dilated aorta, was removed and examined at leisure. The heart was also removed.

Heart. Both ventricles dilated, especially the left, which bulged far into the right. Left ventricle: width, when open, six inches; length, four and one-half inches.

Aorta measured four inches in circumference just above the valves. Two inches above it measured five inches. Aortic valves were thin and normal. Heart muscle was very soft and pale, with considerable post-mortem softening. The tips of the papillary muscle were fibroid. Coronary arteries healthy. Aorta thin and irregularly dilated; it was covered with low atheromatous patches, none of which was recent. The aneurismal sac began at the middle of the arch and continued to the bottom of it. The sac held the hand loosely open, and was a dilatation of the whole arch. There was a distance of two and a half inches between the left carotid and the subclavian. The left lung was adherent to the sac over an area of five by five inches. The adhesions were old and firm, and the lung was spread out in a thin layer over the adherent portion of the sac. The sac was empty; there was no trace of an organized clot. In some spots there were small recent clots, and one of these in the anterior part of the sac where the lung was adherent filled a round cavity, which proved to be a healed ulcer, the base of which was the lung. It was one-fourth of an inch in diameter. Further back was another healed ulcer, which was half an inch in diameter. It was shallower than the other, but showed lung tissue at the bottom.

One inch from the lower end of the sac, on its concavity, was a recent laceration one and a half inches long with irregular edges and somewhat S-shaped. It involved all the coats of the vessel, and opened to the lung. A passage leading from this opening ran down and back between the aorta and lung, and found exit at the beginning of the thoracic aorta. Just beneath the pleura the tract contained a clot about three inches long and half an inch wide. An old and severe rupture of the intima was seen on the convexity, running from the recent laceration toward the subclavian. It was two inches long and one-half to three-fourths of an inch wide. It involved only the intima and was perfectly healed. The adventitia at this spot was strongly reinforced by the adhesions. The thoracic aorta was two and a half inches wide when open. Its surface was wrinkled and showed numerous atheromatous patches, and at the lower end a calcareous plate one-half by one-fourth of an inch in dimension. *May* 12, 1892.

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12. *Malignant endocarditis.*

Exhibited by Dr. H. F. Formad.

The specimen exhibited was a remarkable example of vegetative malignant endocarditis obtained from a case of alcoholism. The man

had died during a protracted spree, and an accurate history was, therefore, not obtainable. The liver was fatty and the kidneys showed the marked passive hyperæmia which I am accustomed to find in alcoholic subjects. *May* 12, 1892.

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13. *Hypertrophied and dilated heart; atrophied liver; contracted kidneys.*

Exhibited by Dr. D. J. M. Miller.

I. D., male, aged 58 years, was admitted into the Incurable Home of the Episcopal Hospital, January 11, 1892. Nothing beyond the facts that his arteries were atheromatous, that he had a systolic murmur at the apex, and that the cardiac area was very large, were noted on admission. On January 20th his urine was examined, but contained nothing abnormal. On the 19th of February he had a severe attack of diarrhœa, for which the resident physician ordered the house astringent mixture, containing tinctura opii in small amount. At five o'clock the next morning he was discovered to be unconscious and rapidly became comatose. As he was passing but little water he was catheterized, and in the scanty urine withdrawn a small amount of albumin was found. The microscope did not reveal casts. The quantity of opium taken being small (but ten minims), and because of the presence of albumin, and from the character of the coma, it was decided the latter was uræmic in nature, and treatment for that condition was instituted, but without avail, death taking place on the evening of the 21st, thirty-six hours after the onset of unconsciousness.

At the *autopsy* a small amount of fluid was found in the right pleural cavity. The lungs were much congested; the heart weighed twenty-nine ounces. The left ventricle wall was one inch in thickness; its cavity was enlarged, containing a few clots. The left auriculo-ventricular opening admitted five fingers; the mitral valve was thickened and apparently incompetent; the aortic valves were thickened, and there were several atheromatous patches just above the sinuses of Valsalva. The right ventricle was slightly enlarged and dilated, but seemed small in comparison with the enormous left chamber. The heart-muscle was pale, and on the inner surface of the left ventricle were several fibroid patches, particularly on some of the papillary muscles. The liver weighed twenty-six ounces, was pale-yellow in color, and somewhat hard on section. The kidneys weighed from five to six ounces; the capsules were adherent, the surface nodular; the cut surface was pale-red, of

tough consistence, and showed marked thinning of the medullary portion. The spleen was enlarged and congested. The arteries were atheromatous throughout.

This seems to be a case of so-called arterio-capillary fibrosis, though no microscopic examination was made to confirm the diagnosis which the clinical symptoms and gross post-mortem appearances seemed to warrant. It shows beautifully the cardiac enlargement consequent upon changes in the arterial system. *April* 14, 1892.

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14. *Dilated right ventricle.*

Exhibited by Dr. Judson Daland.

At the autopsy in this case the heart alone was of interest. The heart was not particularly abnormal, the aortic valves being competent. The right ventricle showed great dilatation, so great that one could almost pass the hand through the auriculo-ventricular orifice. Right ventricle first hypertrophied, followed by dilatation, until regurgitation of the tricuspid followed. The murmur was mistaken for a mitral murmur.

May 26, 1892.

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15. *Atheroma of the aorta; beginning aneurism.*

Exhibited by Dr. Judson Daland.

Case of a man aged 55 years. Considerable change in the aorta. It was highly atheromatous, and just above the valve was a beginning aneurism, which occasioned no symptoms during life except a slow and irregular pulse. *May* 26, 1892.

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16. *Multiple embolism.*

Reported by Dr. G. W. Ziegler.

The case reported was seen thirty-seven years ago. A woman had repeated attacks of asthma, at times very violent, with excruciating pains in the abdomen, but with no vomiting or symptoms of obstruction of the bowels. Morphine temporarily relieved the pains, and the next day all pains in the abdomen had disappeared, but she had severe pains at first

in the right and then in the left foot. Circulation in the popliteal and femoral vessels had stopped. Heat was applied, but the next day gangrene set in. Dr. Atlee was called in consultation, and thought the case to be one of embolism. The next day gangrene attacked the leg, and the patient died soon after.

Post-mortem. Abdomen opened and the aorta opened to the bifurcation, where the right common iliac was found to have been obstructed by a fibrous mass seven inches long, tapering to a point which reached down to the bifurcation of the common, into the external and internal iliac. The theory advanced is that a large clot detached from the heart caused obstruction at the cœliac axis, and remained there some time; then was dislodged, and again became lodged at the bifurcation of the aorta. Dr. Ziegler thought that at an old labor there had been a great amount of hemorrhage, and during the slowing of the heart, due to this hemorrhage, a clot had formed in the heart, which caused the asthma—the asthma being relieved by the dislodging of the heart-clot.

May 26, 1892.

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17. *Extensive aortic-valve disease with fatty heart.*

Exhibited by Dr. Alfred Stengel.

This heart I removed this afternoon from a man, aged about 60 or 65 years, who died suddenly yesterday evening. The history is somewhat as follows: He had been a healthy man all his life until three months ago, having led an active life and used alcohol and tobacco, though not to excess. For many years he was more or less interested in wooded lands in the West, and even until three months ago engaged in active horseback riding and hunting without the slightest inconvenience. During the last three months, however, he has been growing short of breath; has had cardiac pain and palpitations; vertigo and ringing in the ears have been constant. Yesterday he had an attack of great dyspnœa and died suddenly.

The heart weighs twenty ounces; the myocardium is yellowish in color, almost uniformly and, as you see under the microscope, is highly fatty. I have never seen a more advanced stage of fatty degeneration in any disease excepting pernicious anæmia. The mitral valves are thick, but not much affected; the aortic valves are reduced to stumps, which are completely calcified and project into the orifice in such a manner as to occasion stenosis as well as regurgitation. The subvalvular ring is

also calcified, so that when we feel on the outside of the aorta at the orifice we find a rigid calcareous ring within. The coronary arteries are considerably obstructed at their orifices and in their course by atheroma.

The interesting features in this case to me refer to the clinical history. Here is a man who, doubtless, had extensive and destructive aortic-valve and coronary disease for years, who was advanced in years, and who was, in spite of this, able to engage in excesses of the most exacting sort. It argues for a healthy myocardium, and I take it we must regard the high grade of fatty degeneration visible here as a product of the last three months. With a healthy muscular substance we may admit the possibility of exertions such as he subjected himself to causing no symptoms; with a fatty myocardium such would be impossible.

June 9, 1892.

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18. *Aneurism of the heart.*

Exhibited by Dr. W. E. Hughes.

Clinical diagnosis: Aortic aneurism and cardiac hypertrophy in a patient 50 years old, a drinker, and a sufferer from rheumatism. On admission he had marked œdema of legs. Urinary examination was negative. Heart showed signs of great hypertrophy, pulsation strong and heaving, but almost entire absence of heart sounds. Near apex no second sound was heard, but it was accentuated over aortic cartilage. Apparently but one impulse, but near death another developed over the sternum, distinct in position but not in time. Death caused by hemorrhage into bronchial tubes.

Specimen showed aorta to be healthy. A huge aneurism of the left ventricle was found. Pericardial sac almost obliterated. Left ventricle very large and giving an opening to this large aneurism, which seems to have been formed by a myocarditis with partial perforation. It was full of laminated clots. The collar of the aneurism was smooth between the left ventricle and the sac.

June 23, 1892.

19. *Atheroma of heart and cirrhosis of kidneys.*

Exhibited by Dr. E. L. Vansant.

Patient, male, aged 66 years. Clinically the man exhibited repeated attacks of dyspnœa with œdema of the lungs, during which attacks his temperature would rise somewhat above the normal, being normal between the attacks. While under observation he had at least six distinct attacks. Urine showed a quantity of albumin, and at times would exhibit compound granular casts, while other examinations would reveal nothing but albumin.

Autopsy: Heart and kidneys of especial interest. Heart shows thickened valves, with deep fenestration of the right semilunar valve. The aorta is thickened and atheromatous. Walls also thickened.

Right lung was adherent throughout to costal pleura and diaphragm, and some of the bronchial tubes were dilated.

The capsules of the kidneys were thickened and showed old scars. Capsule very adherent in both. In the left one the cortical portion is narrowed and presents here and there minute cysts. Right kidney contained small cysts and one large one about the size of a pigeon's egg. Supra-renal capsules were thickened, indurated, and of a pale-yellow color. I would add that the rapidly-formed œdema of the lungs would disappear readily under treatment, and that there was also some rise of temperature. This is probably the case frequently in chronic nephritis.

Dr. Hughes: The observations of temperature made by Dr. Vansant are of interest in connection with experiments made by Dr. Carter and myself to prove that the temperature in uræmia is probably due to albumoses in the blood. The attacks of œdema in this case cleared up so rapidly because they were uræmic. Many things are uræmic which we are not accustomed to consider as such, and temperature observations should be made more often. Again, there is often high arterial tension ushering in uræmic attacks, and if the high temperature is also determined to be a frequent condition, both would be of great clinical value in order to abort the attacks in their inception.

Dr. Hughes remarked further that the conditions of the arteries which brings on the kidney change is also the cause of the lung condition—emphysema, and, of course, such a lung is more apt to have œdema.

Dr. Vansant: I have collected a number of cases in which the temperature has risen before the attacks come on. It is a question whether

a new poison is formed or whether the deteriorated substance which should be eliminated acts on the heat centres. As to rapid œdema in emphysema, I think that the pathology of emphysema explains this.

June 23, 1892.

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20. *Valvular disease of the heart.*

Exhibited by Dr. Augustus A. Eshner.

The specimen that I present herewith was obtained from a case that had been under the professional care of my friend Dr. C. F. Pettibone, upon whose invitation I was permitted to be present at the autopsy, and from whom I have obtained the clinical history, and permission to make this report.

The patient was a man, C. B. K., aged 47 years, an ice-cream maker. He gave a history of having had mild attacks of subacute rheumatism prior to the age of 23, during none of which he was compelled to take to bed. At the age of 38 he had an attack of acute articular rheumatism, which lasted for about two months, and kept him in bed for three weeks. Four years later he had a second attack of acute articular rheumatism, which kept him abed for two weeks and interfered with his usual occupation for quite two months. At the age of 35 the patient had cough, with free expectoration, and began to lose flesh. Six years ago, and again three years ago, there was slight hæmoptysis. Cough and expectoration were persistent. For four years shortness of breath had been observed, of progressive intensity. To this precordial pain became added, the intensity of the cough moderating. Physical examination disclosed impairment of the pulmonary percussion-note over the apices of both lungs, with harshness of the respiratory murmur and a small number of moist râles. There had never been chills, fever, or sweats. The apex-beat of the heart was displaced to the left of the nipple-line; the area of cardiac percussion-dulness was increased; the carotid vessels presented manifest pulsation. At the apex of the heart and transmitted to the axilla and lower angle of the scapula, and in the aortic area and transmitted in the course of the carotids, could be heard with the systole harsh blowing murmurs.

Three weeks before death the patient had fierce paroxysms of dyspnœa, lasting perhaps for ten minutes and recurring at intervals of ten minutes, in which he gasped for breath, the face becoming cyanotic and the eyes fixed and staring. The dose of the digitalis that had been given was

increased, and the attacks of dyspnœa became less frequent and less pronounced. A week before death thirty drops of digitalis tincture and two drops of a centesimal solution of nitro-glycerin every three hours controlled the paroxysms for six days; the patient could breathe freely and regularly, and could sleep in the recumbent posture. Dyspnœa, however, finally returned and remained present until the final issue.

At the autopsy the body was found emaciated and cyanotic. The abdominal cavity contained a moderate quantity of clear serous fluid; the viscera, except the kidneys (which presented diffuse fibroid changes) appeared to be normal. The left lung was adherent at the apex and posteriorly; the right was free. Both lungs presented small foci of old tuberculosis at the apices; caseation, induration, calcification. The heart was large; its cavities were voluminous and its muscular walls thick. The leaflets of the mitral valve were thickened. The aortic leaflets were greatly thickened and calcareous, and there was a large calcareous mass over one of the coronary orifices.

Dr. Griffith remarked that he had seen the patient six years before, when he first regarded him as a case of beginning pulmonary tuberculosis. The man had a persistent cough accompanied by considerable loss of flesh, and peculiarly suggestive bronchial expiration at the right apex. There was, however, no alteration on percussion; and as the man's condition improved materially under treatment with cod liver oil and hygienic measures, including residence at Cape May, and as the respiratory abnormality at the right apex was not noted some months later, it was thought that the diagnosis of beginning phthisis had been erroneous. In the light of the post-mortem examination, however, we see that there really had been a commencing tubercular trouble, which doubtless became arrested in consequence of the measures instituted. During the early months attention was not drawn to the cardiac condition, nor had he ever any symptoms pointing to failure of this organ. On examination, however, it was found that there was then present distinct to and fro aortic murmur.

Dr. Meigs: Specimens of this kind always call up the question of the way in which crippled hearts become inadequate, and we are forced to the conclusion that the valvular defect itself has not so much of a part in the general pathological progress as has the degeneration of muscular structure, which is here so evident. *September* 22, 1892.

21. *Large aneurism of the thoracic aorta.*

Exhibited by Dr. Augustus A. Eshner.

H. W. G., a compositor, aged 54 years, was admitted to Jefferson Hospital late in the day on September 20th, with a history of having shortly before vomited a large quantity of bright-red blood—more than a pint it was stated. The man was extremely sallow, and rather emaciated. He related that nine months previously he had had an attack of influenza, in the sequence of which his other symptoms seemed to appear. He found that he had some difficulty in swallowing, with a sense of lodgment of ingested food at a point to the left of the ensiform cartilage, to which eructation gave some relief. Flatulent distention occasioned distress. To these manifestations vomiting was added, the rejection of food taking place usually during the act of eating, practically as soon as the act of swallowing was completed. Nausea was wanting, and there had previously never been hæmatemesis. The man had lost forty pounds in weight in the course of nine months. He complained of severe pain in the epigastric region, and of boring pain in the spine at a corresponding level. There was a history of bilious attacks, in which the patient stated that there had been some discoloration of the skin. The bowels were, as a rule, constipated. I saw the man but once during life, and made but a partial examination. On percussion of the abdomen an area of dulness was found within the limits formed by the middle line on the left, the flank on the right, the costal arch above, and a line an inch below the level of the umbilicus, which offered an obscure sense of resistance. The remainder of the abdomen yielded a tympanitic note. The resident physician, Dr. Arnold, had listened carefully to the heart, and had detected no adventitious sound. The pulse was weak. There was no œdema. There was no shortness of breath, and the man maintained the recumbent posture in comfort. There was no cough. There was moderate spitting of blood-mixed saliva. Following the initial hemorrhage the stools had been observed to be tarry. From the apparent cachexia, the emaciation, the dulness on percussion, the sense of resistance on palpation, the situation and character of the pain, and the course and duration of the case, a diagnosis of carcinoma of the stomach was made. On the second day after his admission to the hospital the patient again vomited a considerable quantity of blood and passed some eight ounces by the bowel, syncope following. He failed to respond to stimulants, and died without reacting.

At the *autopsy, held eight hours after death*, the intestines were found moderately, and the stomach considerably, distended, especially toward its cardiac extremity. When opened, the stomach was found to contain about a pint and a half of partly fluid and partly clotted blood. The small and large intestines likewise contained large quantities of dark, semi-liquid blood. The pericardial cavity contained about four ounces of turbid serum and a few flakes of recent lymph. The anterior and posterior surfaces of the heart presented several longitudinal patches of old organized exudation. The ventricles were contracted, the left with particular firmness. The left auriculo-ventricular orifice admitted the tips of three fingers. The mitral leaflets were moderately thickened. The aortic semilunar leafleats presented a similar change, though in much slighter degree. The aorta beyond was rough, irregular, and presented evidences of advanced inflammatory and degenerative changes. The left lung was everywhere bound by adhesions of considerable firmness, and its removal was attended with a good deal of difficulty. The lung was found to have been compressed and to have been much reduced in size. In the course of its removal it was seen to be in intimate relation with a large elastic mass situated in the posterior mediastinum, rather to the left of the median line, behind the pericardial sac, in front of the œsophagus and extending from about the level of the sixth dorsal vertebra to the level of the twelfth dorsal vertebra. The right lung was free, except over a small extent of surface posteriorly and superiorly. The apices of both lungs presented slight puckering on the surface, but on section there was no indication of infiltration of the parenchyma. The bronchial glands were enlarged and anthracotic. The retro-peritoneal glands were slightly enlarged, but firm and elastic. On section they presented a gelatinous appearance. The liver appeared of normal size and texture; its cut surface was pale. The spleen was elongated and flattened. The organ was pale on section, but its gross appearance was otherwise unchanged; its texture was of diminished consistency. The kidneys were, perhaps, slightly reduced in size, and presented numerous cysts upon the surface. On section pallor was evident, while both cortical and medullary tissues seemed to have undergone diminution. The capsules stripped readily, exposing a smooth surface. The bladder contained about four ounces of pale urine. Its walls were thickened. The prostate was considerably enlarged. The thoracic and abdominal viscera were removed *en masse*. The mediastinal mass was proved to be an enormous aneurism of the thoracic aorta that had ruptured into the œsophagus. The aneurism was about seven inches long, about five and a half inches transversely, and four inches antero-

posteriorly. There was considerable thickening, discoloration, and infiltration of the œsophagus about the irregular and ragged opening through which the fatal hemorrhage had taken place. The adjacent portion of the left lung was likewise discolored and infiltrated. The bodies of the vertebræ were not affected. The aneurism was filled almost entirely with a fibrinous, laminated clot of firm, elastic consistence. The lumen of the abdominal aorta, as it came off from the aneurism, corresponded with the circumference of the little finger.

The case here reported presents a number of interesting features. First, as to diagnosis. It is doubtful if this could have been made absolutely by physical examination. The growth was not accessible to palpation, percussion, or auscultation ; but the occurrence of hemorrhage, with the subjective sensation of an obstruction in swallowing on the part of the patient should at least have excited suspicion. The passage of a probang was absolutely contra-indicated. The great size of the aneurism and its unusual situation are also interesting. It is surprising that more pronounced cardiac symptoms were not occasioned, interfering with the action of the heart as the tumor must have done. The development of the aneurism must have been extremely insidious and gradual, to allow of the accommodation of the adjacent structures to the presence of such a large disturbing element, for the patient had pursued his usual avocation until the time of his admission into the hospital.

October 15, 1892.

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22. *Rupture of aorta with hæmatoma.*

Exhibited by Dr. W. B. Jamison.

F. S., female, aged 64 years, was a case of delusional insanity in the Philadelphia Hospital, Insane Department. General health was good, except attacks of indigestion and tube casts in the urine. She became suddenly and seriously prostrated in the night, revived somewhat, to sink into a second attack, which terminated in death before 6 A.M.

At the *autopsy* external inspection revealed a shortening of the left leg of about two inches, due to an old fracture at the hip.

On opening the abdomen the meso-colon and the tissues behind the peritoneum of the right side from the diaphragm to the pelvis were discolored by an extensive hemorrhage. Resting on the bodies of the vertebræ immediately below the liver, a tumor, regular in form, about four inches in each of its diameters, had the head of the pancreas on its sum-

mit and the duodenum curving around the right. Duodenum and pancreas were free.

Thorax. Pleuræ showed slight old adhesions on left and recent adhesions on right side. Lungs both normal and free from congestion or œdema. Pericardium normal.

Heart weight thirteen ounces. All the cavities were filled with fluid blood, black clots, and a small amount of fibrinous clot. Valves all normal. Heart muscle firm, and color good. The first portion of the arch of the aorta showed a vast amount of atheromatous swelling and infiltration, but no ulcers or calcareous infiltration.

Spleen, weighed three and one-half ounces, and was normal.

The left kidney weighed three ounces, and showed well marked interstitial change. The right kidney weighed three and one-half ounces. The lower extremity was stained anteriorly by the subperitoneal hemorrhage. The rest of the organ was not surrounded by the blood. This kidney presented quite as advanced cirrhosis as the other.

Stomach and intestines normal. Pancreas the same. Liver weighed two pounds, and showed atrophic changes.

On removing the tumor and opening the aorta, the intima was found to be everywhere atheromatous, and at the bifurcation there was extensive calcareous infiltration. About two inches above the bifurcation there was an irregular, ragged opening one-half inch in diameter, which looked as if it were recent. After the tumor was allowed to harden in alcohol a free incision showed that the clot was uniform in its appearance and the sac appeared to be composed of the tissues overlying the aorta.

November 10, 1892.

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23. *Aneurism of the arch of the aorta.*

Exhibited by Dr. W. B. Jamison.

John R., aged 53 years, was in the Philadelphia Hospital suffering from a mediastinal tumor. The symptom of greatest importance was the laryngeal disturbance.

At the autopsy nothing noteworthy was found, except in the thorax.

The lungs were both emphysematous. The right lung also showed pronounced congestion and areas of catarrhal pneumonia in the lower lobe.

The heart was normal. An aneurism of the arch was found on the

lower surface of the artery, and showed the left vagus as it crossed the tumor. The dilatation was not large, and did not present any peculiarties. *November* 10, 1892.

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24. *Aneurism of the aorta.*

Exhibited by Dr. M. V. Ball.

There had never been any symptoms in this case. The patient, a prisoner in the Eastern Penitentiary, died suddenly after slight hemorrhage from the mouth. An aneurism was found, which had ruptured into the œsophagus. The cervical vertebræ were decidedly eroded by the aneurism, and organized clots were attached to the eroded bone.

November 10, 1892.

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25. *Chronic rheumatism with deformities—Endocarditis, bovine heart.*

Exhibited by Dr. F. S. Pearce.

James D., aged 11 years, a sufferer from chronic articular rheumatism for five years. Patient had recently been in Polyclinic Hospital, being very low several times from exacerbations of rheumatism and accompanying endocarditis. He was admitted to the Orthopædic Hospital May 19, 1892, under Dr. H. E. Goodman's care, for the deformities of knees and ankles, the legs being firmly flexed upon the thighs, and the feet extended. The knees were swollen, indurated, and firmly ankylosed in the positions stated, while the ankle joints were a little less firmly fixed. The anterior tibial arteries were seen forcibly pulsating between the tendons on front of ankles. Hips ankylosed and pelvis distorted, a large deep and sloughing bedsore being over each great trochanter, caused by the curled-up position he lay in, and resting upon these bony prominences. Patient was quite anæmic, entire surface of body being waxy, and most all joints swollen, and painful on attempted motion, although there was but little elevation of temperature above the normal. He was ordered massage and gradual extension at the knees by splints, the syrup of the iodide of iron being given internally with alkalies.

On June 4th he had an acute exacerbation with temperature up to 102°; considerable dyspnœa, subcrepitant râles heard at both bases of

the lungs, slight impairment of resonance, and increased fremitus. Heart sounds weak, percussion showing diplacement to one-half inch to right of sternum, the cardial dulness being also from third costal cartilage down to sixth interspace in line of nipple. Systolic murmur at apex, and aortic cartilage. Heaving impulse and drawing in of apex at beats. Loud pericardial friction-sounds also heard synchronous with heart beats. Urine loaded with uric acid.

From June 6th to 23d, temperature ranged from 102° to 104°, pulse from 115 to 130, and irregular, while the respirations numbered as high as 40, and were irregular and shallow. Patient restless, cannot take food, emaciating rapidly, and temperature falling to 100°, where it remained with patient pulseless for several days before death, which took place July 3, 1892, of heart failure, the temperature being 108.5° one-half hour before death.

Autopsy. Body of an emaciated boy, much flexed, and contorted limbs.

On opening the thorax both pleural cavities were entirely obliterated by dense fibrous adhesions, which were with difficulty separated.

Heart very large (bovine)—six and a half inches from base to apex—and prominent, pushing left lung up into the clavicular region, compressing it into half of its normal size. The right lung was also small, both of them being deeply congested. No tubercles.

Pericardial layers glued together by firm and old fibrous adhesions in front, while posteriorly the sac was glistening. Vessels severed, and heart removed with part of trachea and thymus gland intact. Left ventricle distended by currant-jelly clot.

Heart muscle firm, slight vegetations on aortic, semilunar, and mitral valves.

Right auricular walls very thin. Heart is interesting as a specimen of extreme hypertrophy in a child. Kidneys normal.

December 8, 1892.

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26. *Remarks on the pathology of the blood.*

Read by Dr. Frederick P. Henry.

Our only criteria of disease are alterations of structure and function, but in this Society the term "pathological" is necessarily limited to the former. My remarks will, therefore, be chiefly confined to certain alterations in the visible constituents of the blood which we have learned, by

experience, to associate with definite clinical phenomena. Incidentally, I will refer to certain structural changes in various tissues which are either manifestly or presumably secondary to those of the blood.

The diseases which I shall briefly discuss are comprehended under the generic term "anæmia," which signifies a diminution in the number or value of the red blood-corpuscles. We recognize primary and secondary anæmias, or, as some prefer to call them, idiopathic and symptomatic anæmias. My remarks refer solely to the primary forms of anæmia.

By a primary anæmia I understand an affection in which the initial lesion is seated in the blood itself, or in one of the organs, universally recognized by hæmatologists as hæmatopoietic or blood making. These organs are the spleen, the lymphatic glands, and the bone marrow.

The type of primary anæmias is chlorosis, because, in this disease, the primary changes are those of the blood, all others being secondary to the alterations in this fluid. According to many hæmatologists, pernicious anæmia is entitled to the same distinction, but I will endeavor to prove that this claim has not as yet been substantiated.

Besides chlorosis and pernicious anæmia, the primary anæmias include the various forms of leucocythæmia, lymphatic anæmia or Hodgkin's disease, and splenic anæmia.

Many years ago, when the study of blood diseases was wofully lacking in precision, the term pseudo-leukæmia was introduced and applied to cases in which, with a normal number of white blood-cells, the lesions were identical with those of leucocythæmia. In my opinion this term should be discarded as one serving merely to complicate an already intricate subject. An affection of the blood is either leukæmic or non-leukæmic. We are quite as well justified in speaking of pseudo-pneumonia or pseudo-scarlatina as of pseudo-leukæmia.

I will discuss very briefly the pathological anatomy of the primary anæmias in the following order: (1) Chlorosis; (2) Pernicious anæmia; (3) Leucocythæmia; (4) Lymphatic Anæmia or Hodgkin's Disease; (5) Splenic Anæmia.

Chlorosis. Virchow endeavored to place chlorosis upon a distinct anatomical basis by the demonstration that, in fatal cases of this disease there is often found an imperfect development of the aorta and arterial system generally. He has found the aorta of a full-grown woman so small as barely to admit the little finger, whereas, normally, it should admit the thumb; and with this contraction of the lumen of the vessel its coats were observed to be much thinner than normal. He regarded this condition of the vessels as congenital, and the importance of the observation depends chiefly upon the fact that the bloodvessels and the

blood corpuscles are both derived from the same embryonic layer, the mesoblast; an imperfect evolution of the one probably entailing the same condition in the other. In short, Virchow regards chlorosis as due to a congenital predisposition to anæmia. I have no doubt that this may be true of such cases as are distinguished by an obstinate tendency to relapse, as well as of those that run a fatal course. Such a condition as that described by Virchow—a condition of imperfect development of the vascular system—would doubtless give rise to grave disturbances of nutrition which might lead to a fatal issue, but chlorosis is not, as a rule, a fatal disease, the great majority of cases, under appropriate treatment, ending in recovery; and with reference to these cases there is no proof of a stunted state of the bloodvessels. The only constant anatomical characters of chlorosis are those of the blood itself, and even these changes are not uniform. The researches of Duncan, in 1867, first established the fact that, in well-marked cases of chlorosis, the number of red corpuscles might be normal while their percentage of hæmoglobin was greatly reduced, and this anomaly was for a long time regarded as the distinguishing mark of chlorosis. It has since been established that this view of the blood change in chlorosis is altogether too narrow, and, at the present day, it is well known to those who have studied this question that, in chlorosis, the changes in the blood may be at least threefold:

(1) The blood corpuscles may be of normal size and number, their only change being a deficiency of coloring matter; (2) they may be diminished in size and normal in number and in percentage of coloring matter; (3) they may be diminished in number with diminished, normal or, perhaps, increased percentage of hæmoglobin.

From the above facts it is manifest that there is nothing uniform in the blood changes of chlorosis, so that an attempt to classify it as a distinct disease from the standpoint of the blood changes must result in failure. The essential fact is that the percentage of hæmoglobin is reduced in this affection, but this is common to many other forms of anæmia. It has been stated by Immermann that chlorosis differs from all other forms of anæmia in that, while the red corpuscles may present the various changes above mentioned, the albuminous substances of the serum are present in that fluid in normal or increased amount. This has certainly been proved to be true in a few cases by chemical examination, but it has not yet been shown that the same may be true of other forms of anæmia.

Turning from the blood to the other organs of the body we find the same want of uniformity in the changes which they present. The heart has been found abnormally small in some cases, abnormally large in

others, while the organs of the genital system, which are popularly supposed to be particularly affected in chlorosis, have been found imperfectly developed in some cases, while in others their development has exceeded the average normal limits.

I have elsewhere defined an independent disease to be one which rests upon a constant anatomical basis, or is invariably produced by the same specific agent.[1] If this definition is correct, chlorosis cannot be regarded as an independent disease. I admit that the definition to which I refer is an incomplete one. Much may be said in favor of the view that a uniform *ensemble* of symptoms may constitute a disease, apart from uniform lesions, but in this Society our discussions are limited to lesions.

Pernicious Anæmia: The blood changes of pernicious anæmia are more profound than those of any other disease. In the first place, the quantity of the blood is so much diminished that it is often with great difficulty that the requisite amount—a drop or two—is obtained from the finger for examination. Even the gross appearance of the blood as it flows from the puncture is quite diagnostic. It is of a watery consistence, and of a pale pink color tinged with yellow. Its appearance has been aptly compared to that of water in which beef has been washed. On counting the corpuscles their number is found to be extraordinarily reduced. To give an idea of the diminution of the red corpuscles in pernicious anæmia, I will compare the state of the blood in this disease with that found in cases of cancer of the stomach. I know of no disease outside of the so-called blood diseases in which the "*cachæmia*" is so profound as in gastric carcinoma, and yet the number of red corpuscles in that affection never reaches so low a point as is commonly attained in pernicious anæmia.

In a case of cancer of the pyloric end of the stomach in which I examined the blood on February 7, 1888, I found the number of the red corpuscles to be 2,400,000; *i. e.*, 48 per cent. of the normal; whereas, in pernicious anæmia I have found 525,000 (10.5 per cent.) in one count; 560,000 (11.2 per cent.) in another; and 315,000 (6.3 per cent.) in a third. There is no other affection in which the number of red corpuscles is reduced to figures which can be compared with these. With this extraordinary reduction in the number of these bodies there goes an increase of their size, many of them being double the size of the normal red corpuscles. This increased size of the red corpuscles might, perhaps, be regarded as a conservative provision of Nature by which they are enabled to carry more hæmoglobin than would be contained in an equal

[1] Transactions Association of American Physicians, vol. iv.

number of corpuscles of normal size. Be this as it may, it is certainly true that, in the blood of pernicious anæmia there is often a larger percentage of coloring matter than of red corpuscles. This fact, however, is sometimes susceptible of another explanation. The blood of pernicious anæmia often contains immense numbers of minute bodies resembling "small red-tinged fat globules," to which attention has been particularly directed by Eichhorst. These minute bodies are not counted among the red corpuscles, but undoubtedly contribute toward increasing the percentage of coloring matter.

Another change undergone by the red corpuscles in this disease is alteration of shape—"poikilocytosis." This change is a constant one, but is not peculiar to pernicious anæmia. I have recently seen it in a typical case of chlorosis, and it has been observed in cases of cirrhosis of the liver and in other affections.

The other anatomical alterations of this disease are secondary to those of the blood, and consist principally of fatty degeneration, hemorrhage, and pigmentation. The fatty change is most marked in the heart, especially in the left ventricle, and is particularly prominent in the papillary muscles. It occurs in the form of zigzag lines easily seen by the naked eye, which are known as "tabby mottling," a name given them by Dr. Quain. Fatty changes may also be observed in the lining membrane of the arteries and in the cells of the capillary vessels.

The most constant seat of hemorrhage is the retina, and this fact is one of great diagnostic significance. It is said that the hemorrhagic spots are much more numerous, as a rule, and smaller than the extravasation of albuminuric retinitis. After the retina, the most frequent seats of hemorrhage are the membranes of the brain and the serous membranes generally, and the mucous membranes of stomach and lungs. Petechiæ and ecchymoses of the skin are rare.

The third principal secondary change to which I referred is pigmentation. It is undoubtedly the most important of all the anatomical alterations of pernicious anæmia, and for this very reason I will postpone its consideration until the last.

It was suggested by Prof. Pepper in 1875 that the origin of pernicious anæmia might be traced to certain changes in the marrow of the bones, which virtually consist in a return of this tissue to its fœtal condition. This view of the pathology of the disease attracted considerable attention, but it has since been shown that in many cases the marrow is perfectly normal. It is now believed that the change occasionally observed in the marrow are secondary to the altered state of the blood, for

they have been observed in other diseases attended with great anæmia, such as carcinoma of uterus, rectum, and breast.

It is, I think, highly probable that important changes have often been overlooked in the bodies of those dying with the symptoms of pernicious anæmia. For example, in a case reported by Prof. Osler and myself,[1] in which every symptom of pernicious anæmia, including retinal hemorrhages, was present, we found, at the autopsy, an atrophy of the mucous membrane of the stomach so extensive that not a single normal tube could be found. To a practiced eye even without the microscope, it was evident that a certain amount of atrophy was present, but no idea of its extent could have been obtained without a thorough microscopic examination.

Lichtheim[2] has recently called attention to the frequent occurrence of spinal degenerations, especially of the posterior columns, in cases of pernicious anæmia. "The symptoms of this condition are masked by others dependent upon the profound anæmia, so that attention is rarely directed to them. In none of the cases examined—their number is not stated—was the cord found in a normal condition. In one-half of them the only morbid change consisted of small submiliary, sclerotic foci, shown by the microscope to have their origin in apoplectic extravasations. In the other half there was widespread degeneration of the posterior columns. The columns of Clarke and the posterior nerve-roots were never involved. The condition is undoubtedly secondary, precisely similar changes having been observed by Lichtheim in a case of splenic leukæmia."[3]

These interesting changes were, I believe, first detected in this country in a case under the care of Dr. Morris J. Lewis. The microscopic examination was made by our fellow-member, Dr. Charles W. Burr, who has in his possession several slides from that and other cases which clearly illustrate this spinal lesion.

The adipose tissue of those who have died in pernicious anæmia is often found of a deeper, richer yellow than normal. The muscles, also, are generally of a deep red color, and the abdominal organs, especially the liver, are sometimes decidedly darker than under normal circumstances. These facts, especially the dark color of the abdominal viscera, have been used to support the theory that the cause of pernicious anæmia is to be found in an increased destruction of the red corpuscles, for, as is well known, all the pigments of the economy are derived from these bodies.

[1] American Journal of the Medical Sciences, April, 1886.
[2] Berliner klin. Wochenschr., October 14, 1889.
[3] F. P. Henry, in Sajous' Annual of the Universal Medical Sciences, vol. ii, 1890.

In 1876 Quincke demonstrated a great excess of iron in the liver of those who had died of pernicious anæmia, and this observation was confirmed by Rosenstein and Peters. It is probable that the dark color of the muscles, and the deep yellow color of the adipose tissues in this disease are due to an excess of iron. It was at first objected that the iron in the liver was derived from that administered to the patient, but it has been clearly proved that this is not the case. The amount of iron in the liver appears to be practically uninfluenced by the medicinal administration of this substance.

Quincke's observations do not seem to have attracted the attention they deserve; but this was doubtless, in part, due to the fact that he was not positive in his opinions as to the source of the iron. He thought it might have been derived from preparations of that metal administered to the patient.

In 1888, Dr. W. Hunter, in a series of articles in the *British Medical Journal*,[1] not only confirmed Quincke's discovery, but showed that the iron in the liver is deposited in a special manner, being contained, for the most part, in the liver cells, and in those of the outer and middle zones of the acini. The central cells are generally in a state of fatty degeneration and atrophy, and often fail to give any iron reaction.

It is impossible, in the limits of this paper, to discuss the careful steps by which Hunter arrived at the conclusion that pernicious anæmia is a special hæmolytic process. It must suffice here to quote the last of his "summary of results:" "In pernicious anæmia the seat of disintegration is chiefly the portal circulation, more especially that portion of it contained within the spleen and liver, and the destruction is effected by the action of certain poisonous agents, probably of a cadaveric nature, absorbed from the intestinal tract."

Hunter has also called attention to the dark color of the urine in pernicious anæmia, and has proved it to be due to the presence of pathological urobilin, a substance known to be "derived from the disintegration of hæmoglobin." I observed an unusually dark color of the urine in a case of pernicious anæmia several years before Hunter's publication, but did not ascertain its cause. It struck me simply as a remarkable fact that with so few red corpuscles (between one and two million to the cubic millimetre) the urine should be so deeply colored.

The case in question was a gentleman, a patient of Dr. Weir Mitchell, whose blood I examined on several occasions. I called Dr. Mitchell's attention to the appearance of the urine, and he, like myself, was struck with it.

[1] September 22d, September 29th, October 16, 1888.

The proofs in favor of the view that pernicious anæmia is associated with increased hæmolysis are certainly convincing. Hunter's observations have been confirmed from various sources, and especially in Philadelphia, by Drs. Griffith and Burr,[1] in a paper distinguished by close thinking and careful research. I accept their conclusions, and those of Hunter which they confirm, that pernicious anæmia is distinguished by increased hæmolysis, but I think it is going too far to say that it presents no evidence of imperfect hæmogenesis.

I do not see how such capable observers as Griffith and Burr can deny the evidences of imperfect hæmogenesis in pernicious anæmia. The large, flabby, misshapen red corpuscles of pernicious anæmia certainly present anything but a healthy aspect. It cannot surely be argued that these remarkable appearances are the result of a struggle to escape destruction while running the gantlet of the liver.

In addition, numerous nucleated red corpuscles are often present in the blood of pernicious anæmia, and everyone acknowledges such corpuscles to be embryonic.

Again, the red corpuscles of pernicious anæmia are often possessed of amæboid movement, a fact to which attention has been called by Hayem,[2] and which I have myself observed. This property is characteristic of the lower order of cells, the leukocytes.

Finally, Rindfleisch, who has made a careful study of the marrow in two cases of pernicious anæmia, concludes that the disease is due to defective hæmogenesis. He bases this opinion upon the absence of elements which he and others regard as transition forms between the red nucleated marrow cells, which were unusually large and abundant in his cases, and the fully developed red corpuscle.[3]

In view of the above-mentioned facts, it seemed to me that the evidences of defective hæmogenesis in pernicious anæmia are, at least, as strong as those of increased hæmolysis.

Leukocythæmia. My remarks upon the remaining forms of primary anæmia must be of necessity brief and imperfect.

In leukocythæmia the blood change never exists independently, but is always associated with anatomical alterations of spleen, lymph glands, or bone-marrow. Accordingly, three forms of leukocythæmia are commonly spoken of: a splenic, a lymphatic, and a myelogenic. The splenic form is, as its name implies, associated with disease of the spleen, and, according to some authorities, this is the only genuine form

[1] Medical News, October 17, 1891. [2] La Semaine Médicale, February 26, 1890.

[3] Vide Münchener med. Wochenschrift, June 3, 1890; also Virchow's Archiv, Bd. cxxi, 1890, p. 17.

of leukocythæmia, the changes sometimes observed in lymph glands and bone-marrow being, in their opinion, secondary to those of the blood.

The spleen is often enormously enlarged, and may extend downward into the false pelvis, and forward as far as, or even beyond, the median line. It has been more than once mistaken for an ovarian tumor. Its peritoneal covering is often the seat of inflammation which gives rise to marked thickenings and opacities and adhesions to neighboring organs. The tissue of the organ is much firmer than normal, and its cut surface is smooth, closely resembling the cut surface of the liver. Hemorrhagic infarctions are often found in the substance of the organ. Under the microscope no changes except those of simple hypertrophy can be detected.

In a certain number of cases, it is said (by Fagge) in about one in every three, the lymph glands are enlarged, and here also under the microscope no changes but those of simple hypertrophy are present. Their consistence is generally soft, sometimes almost fluctuating, and their color white, yellow, gray; or, as in the case of the bronchial glands, dark purple, or black, from pigmentary deposit.

The changes in the marrow are analogous to those in the spleen and lymph glands, and consist of an enormous accumulation of leukocytes, which gives to the tissue a purulent appearance.

Alterations in other than lymphatic organs are secondary to the condition of the blood, and consist in the formation of lymphatic or adenoid tissue, either as a diffuse infiltration or in nodular masses resembling miliary tubercles. The liver and kidneys are the organs chiefly affected, and the former may be so greatly enlarged as to reach the crest of the ilium. The peritoneum may be covered with small nodules which, under the microscope, are seen to possess the same structure as the lymph glands, viz., a reticular tissue enclosing leukocytes in its meshes. These same deposits of lymphatic tissue have also been observed in the retina, in which tissue, also, hemorrhages are quite as common as in pernicious anæmia. Retinal hemorrhages were observed by Gowers in four out of five cases.

The essential anatomical alteration in this disease is the increase, sometimes enormous, in the number of the white blood-cells. The red are correspondingly reduced, but their number is not so small as in pernicious anæmia, being seldom lower than 50 per cent. of the normal. With reference to its volume, the blood of leukocythæmia is in marked contrast to that of pernicious anæmia. In the latter disease the volume of the blood is so reduced that it is often a matter of difficulty to obtain a drop from the finger for microscopic examination. In leukocythæmia,

on the other hand, the vessels are not only well filled, but there is sometimes a state of increased vascular tension. After death, the cavities of the heart have been found dilated and the vessels abnormally filled with blood, so that the condition of the blood in this disease has been termed "leukæmic plethora."

I leave to Dr. Stengel, the co-referee in this discussion, the description of the minute changes in the white corpuscles, which only become apparent when these bodies are stained by certain well-known methods.

No well-marked case of leukocythæmia can escape detection if the blood is properly examined. It is evident at a glance that the white cells, as a whole, are abnormally increased in number; but it is quite another thing to determine the relative share of the different varieties of white cells in this increase. It is well known that there are, in normal blood, various kinds of white cells, which may be classified either morphologically, or in accordance with their reactions to various staining substances. Morphologically, we recognize small and large lymphocytes, both of which are mononuclear, polynuclear leukocytes, and mononuclear transition forms. According to the second method of classification, the white cells are divided with reference to the affinity displayed by their protoplasmic granulations for certain coloring matters. The latter is the classification generally adopted by hæmatologists, whose leader in this line of research is, confessedly, Ehrlich. Owing to the researches of this observer, an entirely new nomenclature has arisen, and we now speak of cells that are eosinophile, basophile, neutrophile, and amphophile. These terms are largely self-explanatory, but their precise significance will appear in the course of Dr. Stengel's demonstrations.

In normal blood the great majority of the white cells are neutrophile, whereas in leukocythæmia the basophile and eosinophile cells are often very abundant. It was at one time supposed by Ehrlich that an increase in the number of the eosinophile cells was a characteristic sign of leukæmia, and that by it the diagnosis between leukocytosis and leukocythæmia could be readily made in doubtful cases. This theory has not been confirmed, and Ehrlich himself has abandoned it. In individuals free from leukæmia an increase in the eosinophile cells has been observed and, on the other hand, in well-marked cases of leukæmia the eosinophile cells may be absolutely but not relatively increased; in other words, the leukæmic state of the blood may be chiefly due to the increase of other elements.

The leukæmic blood change is characterized not only by excess of elements normally existing in the blood, but by the appearance of a cell not seen under any other circumstances. This is the so-called myelocyte.

It is as large as, or even larger than, the large lymphocytes of normal blood, and differs from the latter in that it contains a large number of neutrophilic protoplasmic granules. It is, I believe, undoubtedly derived from the bone-marrow. This opinion is chiefly based upon its resemblance to cells found in that tissue, but there is further evidence in its support. In a case reported many years ago by Mosler, these myelocytes were found in the blood. In the sternum were two spots upon which pressure gave more pain than elsewhere: it is described as intense. The spots were soft and elastic, and the sensation they communicated to the finger when pressed firmly into them was that of a cavity with sharp edges. During the life of the patient Mosler withdrew from one of these cavities, by means of a trocar, some of its contents. They contained, besides a small number of red globules and fat drops, a large number of cells identical with those found in the blood.

Another ingredient of leukæmic blood is the pointed octahedral crystal discovered by Charcot in 1853. It is identical with the crystal found in asthmatic sputum by Leyden, and, according to Neumann, is a normal ingredient of the bone-marrow. It appears to be associated with the retrograde metamorphosis of leukocytes. I am not aware that this crystal has been heretofore found in other than leukæmic blood, but I have in my possession a slide containing a preparation of blood from a well-marked case of pernicious anæmia in which these crystals are abundant. In the case to which I refer the white cells were not increased.

It was my intention to say a few words concerning the pathology of the disease variously known as Hodgkin's disease, adenia, pseudo-leukæmia, malignant lymphoma, etc., but this paper has already attained an inordinate length. Besides, I must confess my inability to draw a strict line of demarcation between this disease and lymphatic leukæmia. I have elsewhere given my reasons for this opinion.[1]

Concerning the rare disease called splenic anæmia, very little is known. By this term I understand an affection with all the clinical features of pernicious anæmia, and with great enlargement of the spleen. I saw such a case several years ago at the Pennsylvania Hospital, the patient being under the care of the late Dr. James H. Hutchinson. The characters of the blood were identical with those of pernicious anæmia, and the spleen was greatly enlarged. At the autopsy, at which I was not present, the spleen was found dark and diffluent, like the currant-jelly

[1] Transactions of the Association of American Physicians, vol. iv.; also Medical News, October 5 and 12, 1889.

spleen of typhoid fever. This affection has been particularly studied by Banti,[1] of Italy, and Strümpell,[2] of Germany. The accounts of the condition of the spleen are somewhat conflicting. Those who retain the term pseudo leukæmia would readily solve all the difficulties concerning the semeiological status of this disease by calling it the splenic form of pseudo-leukæmia.

In conclusion, I would say that I am fully aware of the deficiencies of this paper. The subject is too vast for one evening's discussion. I wish also to express my gratification at being selected as the spokesman of the numerous members of this Society who are specially interested in the diseases of the blood, and who have advanced its study by careful clinical and pathological work. *December* 22, 1892.

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27. *The methods of examining the blood.*

Read by Dr. Alfred Stengel.

I was asked to read a paper on the methods of examining the blood, but as this would of necessity be a very difficult matter if I did not refer at all to the structural conditions, it has been arranged by Dr. Henry and myself that I should extend my observations to the consideration of the histology of the blood as well.

The subject may be divided into—first, the study of the physical and chemical properties of the blood and the estimation of the number of corpuscles; and, second, the minute examination with the aid of the microscope.

It is needless for me to detail at length the methods of counting blood corpuscles, or of estimating the hæmoglobin, both of which procedures are well known to you, but there are certain points to which I wish to call attention.

The red corpuscles are now generally counted by means of the instrument of Thoma-Zeiss, and, I think, with good reason. This apparatus has distinct advantages over those of Hayem, Gowers, or Malassez in one or other of the following points—the simplicity, the compactness, and the advantageous division of the squares on the slide. The division into small squares, sixteen to the large square, is an immense advantage over the oblong divisions of other instruments, in which the size of the spaces gives rise to confusion and great eye-strain. In regard to the

[1] Annali Universali, Parte rivista, 1883.

[2] Archiv der Heilkunde, vol. xviii., 1877.

diluting fluid, I wish merely to say that of the many which I have employed from time to time none has given such satisfactory results as the 2½ per cent. solution of potassium bichromate, which Daland has so highly lauded. I have used it continuously for over a year, and find that it fixes the corpuscles at once and permanently, without change of shape or appearance, and without any danger of small conglomerations or coagula, and that the solution keeps indefinitely without change. One other point I think worth speaking of. I have frequently found the difficulty, when I have tried to draw the blood only to the 0.5 mark, that it would overshoot this mark by capillarity; whereas this difficulty is rarely encountered at the 1 mark owing to the sudden expansion of the tube immediately above. If the blood does rise above the mark, and is then expelled down to it, so many corpuscles cling to the side of the tube as to vitiate the count. The difficulty I speak of is especially apt to occur when the pipette is not thoroughly clean and dry, but may happen in any case.

Blix first introduced the principle of centrifugal force in the estimation of the number of corpuscles in his hæmatokrit, and Daland, among others, advocates the use of this instrument. The blood, diluted with an equal quantity of Müller's fluid or solution of bichromate of potash, is drawn into a capillary tube 35 mm. long and 1 mm. in diameter, which is then placed horizontally on an arm of the instrument, which may be made to revolve with great rapidity. The heavy red corpuscles find their way to the distal end in a few minutes, and by reading off the number of graduated spaces filled, the absolute number of corpuscles per cubic millimetre is readily calculated by means of a factor experimentally determined to be about 100,000. Thus, if the red corpuscles fill five spaces, there are 500,000 corpuscles, etc. The advantages of this instrument are the rapidity with which the count is made, the relief of the eyes from the strain of microscopic work, and the accuracy of the results, which I believe are as reliable as those obtained even by the Thoma-Zeiss instrument. The disadvantage is the impossibility of estimating the number of leukocytes, but certain alterations may perhaps obviate this difficulty.

A special form of the Thoma-Zeiss instrument has been made for counting white corpuscles. It differs from the ordinary one only in that the blood is diluted with but nine parts of fluid, so that a much greater number of leukocytes is present in a given quantity of the mixture. The best diluting fluid to use is a 3 per cent. acetic acid solution, which disintegrates and removes from view the red corpuscles. In practical work I see no use for this instrument, because, with leukocytosis of appreciable

degree, the ordinary instrument suffices, and when leukocytosis is not present the number of white corpuscles may be disregarded.

The counting of blood plaques has sometimes been encouraged, but thus far no practical lessons have been learned. Normally there are from 200,000 to 500,000 per c.mm. Affanasiew and Fusari have noticed increased numbers of plaques during the period of regeneration of the blood in apyretic anæmias, and I am able to confirm this statement from my own observation. The difficulty in counting these bodies is their extreme liability to stick together and to disintegrate. A modified form of Flemming's solution has been recommended by Pruss, and a saline solution to which 0.6 per cent. of peptone has been added, by Affanasiew, as diluting fluids.

Two forms of instruments have been devised for estimating the hæmoglobin. In the first form the thickness of the layer of undiluted blood through which lines or figures may be seen indicates the quantity of hæmoglobin; and in the second form, blood diluted with distilled water is compared with standard colors. The former principle is employed in Henocque's instrument, but this has such grave disadvantages that I think we may say it is practically useless. The two instruments which depend upon the latter principle, and which divide honors, are that of Gowers and that of Fleischl. Dr. Henry formerly used Gowers' apparatus and preferred it, but has more recently employed the Fleischl. Personally, I like the latter, and this seems to be more and more favorably received, but it has certain disadvantages. In the first place it is necessary to use artificial light; and in the second place, in the scale, which runs from 0 to 100 and more per cent., the 100 per cent. is supposed to be normal, whereas, as a matter of personal experience, I may say there are very few healthy men who exceed 85 per cent. This error has been particularly studied by Dehio, who found the reading constantly in excess, but especially so when the quantity was small. Only once in a large number of cases have I seen a percentage as high as 100. This was in the person of a plethoric individual whose corpuscles numbered 6,000,000 per c.mm., and whose hæmoglobin was one time 105 per cent. and again 95 per cent. A practical point of great importance, and too frequently disregarded, is the avoidance of congestion of the finger when the blood is drawn. This might seem on general principles to make little difference, but the influence of peripheral congestion was well seen in one of my cases—a case of profound cardiac anæmia, in which, during an attack of general cyanosis from cardiac hyposystole, the percentage of hæmoglobin rose fully 15 per cent. This point was also well shown by Winternitz, who found the blood of the toe to contain

15 per cent. more hæmoglobin than that of the finger held above the head.

There remain to be considered certain examinations of less practical value, but which may be of interest in certain cases. The quality of the coloring matter of the blood is easily and definitely determined by spectroscopic examination, and in hæmoglobinæmia the same test may be applied to the plasma obtained after sedimentation of the corpuscles, the blood being kept at a low temperature. Practically, there is little necessity of this examination for hæmoglobinæmia, as this condition in even mild degrees may lead to hæmoglobinuria or other visible signs, and in severe cases the corpuscles are seen under the microscope floating in a pinkish plasma.

The specific gravity of the blood in health is about 1055, but varies greatly with varying conditions of diet, etc., and in disease bears no constant relation to any form, excepting that, as a rule, the density is diminished in severe anæmias. The best mode of estimation, it seems to me, is the modification of Roy's method practised by Lloyd Jones and Monckton. A capillary tube is bent at a right angle near one end; the blood is drawn into this, and then a drop expelled in mixtures of glycerin and water of different specific gravities. By noting in which mixture the drop of blood neither rises nor sinks, but moves horizontally from the end of the tube, and by taking the specific gravity of the mixture, the density of the blood is easily determined. More accurate methods have been suggested, but they are less readily performed.

The alkalinity of the blood has seemed to some a matter of importance. It is best determined quantitatively by titrating with standard solutions of acetic (Lassar) or phosphoric acids (Zuntz), and testing the blood from time to time by placing a drop on a plaster-of-Paris plate impregnated with neutral litmus, and quickly washing off the drop with distilled water, when the blue or red color proclaims alkalinity or acidity, as the test proceeds.

These are the main tests of the general and chemical properties of the blood, but with the exception of the numeration of the red corpuscles, and the estimation of the hæmoglobin, they are of far less value than the microscopic investigations I am about to describe. The latter may be considered under two heads: the simple examination of a drop of fresh blood under a cover-glass, and the examination of fixed preparations.

Examination of fresh preparations. A small drop of blood issuing from a prick of the finger is touched on its summit with a cover-glass, which is quickly placed upon a slide. The weight of the cover-glass or, per-

haps, the slightest pressure suffices to spread it evenly and in a thin layer.

The various forms of red corpuscles—microcytes, macrocytes, and poikilocytes—are readily seen. Each of these forms has deservedly attracted careful attention, but much remains to be learned of their production and significance. The diagnostic importance ascribed to the poikilocyte by Quincke can no longer be entertained, and there seems no little probability that Maragliano's view, that they are evidence of rapid degenerative processes, is a correct one. Occasionally in pernicious anæmia we may see red corpuscles which present distinct amœboid movements, to which Hayem particularly has called attention, and further, he has described certain forms which assume a rod shape or irregular contour, and by their shape and active motion well deserve the name "pseudo-parasites" which Hayem has proposed. I have seen these structures several times, and can attest to their deceptive appearance. In the blood of the fœtus, as well as children up to the third month (Luzet), and in that of various grave anæmias, red corpuscles containing nuclei are often detected, though their certain recognition demands the use of stains.

Definite degenerative appearances have been described by Foà, Ehrlich, and Maragliano. Foà demonstrated certain granular appearances by means of staining with methyl-blue; Ehrlich described a reticulated appearance of the stroma, and more recently a separation of hæmoglobin from the other portions, and Maragliano has investigated the degeneration and disintegration of corpuscles under the influence of globulicidal agents. My own observations have disclosed the presence in advanced anæmias, either chlorotic or of other kinds, of corpuscles containing sometimes indefinite granular matters, at other times definite round globules or granules of high refraction and distinct pinkish color, and which suggested the idea of separated masses of hæmoglobin. My first impression, however, on seeing these appearances, was to consider them artefacts, but repeated and careful examinations have convinced me that they are really alterations of the structure of the corpuscle. I cannot say that the corpuscles have seemed generally paler, nor have I been able to determine the nature of these bodies by staining the fixed preparations. They have been most abundant in the blood of cases of pronounced chlorosis, and though visible in ordinary fresh preparations, have seemed especially marked in blood diluted with solution of bichromate of potash.

The white corpuscles are seen in the fresh blood as granular, amœboid bodies, whose nucleus may or may not be distinguishable. They are

easily separated into the large and small forms, but their more exact study requires fixed and stained preparations. Cavafy long ago pointed out that the amœboid motions are less active in leukæmia, but this observation conveys only half the truth and an erroneous impression. There is no diminished activity of the leukocytes in this disease, but there is great increase in the number of lymphocytes or mononuclear forms, which normally are sluggish in this respect. In cases of great destruction of red corpuscles, as in malaria, the leukocytes may contain pigment matters which they have appropriated by reason of their phagocytic property, and in the same way various substances may be included. I have a very beautiful preparation of frog's blood in which one of the crescentic forms of the parasite *Drepanidium ranarum* is seen enclosed in an eosinophilous leukocyte, and a similar condition has frequently been noted by various observers with malarial organisms in human blood.

Associated with the increased number of leukocytes, and doubtless dependent on this, there may be seen in leukæmic blood the peculiar octahedral crystals of Charcot and Leyden. A few observers have seen them in the fresh leukæmic blood, but they increase in number when the preparation is kept some time and in the blood after death, both of which facts indicate their nature as degeneration products.

In the same connection I may speak of the fibrin filaments to which Hayem and the French writers generally have directed attention. These may be seen as delicate threads interlacing and entangling the corpuscles in their meshes. Lilienfeld describes them as passing from masses of blood plaques to the nuclei of adjacent leukocytes; certainly blood plaques seem to play no little part in their production. They are readily seen in a drop of blood kept for some time under the cover-glass, and are numerous in certain acute diseases in which clotting is active, such as pneumonia and rheumatism. No special value can be ascribed to their occurrence.

The blood plaques may be seen in fresh preparations in small clumps closely associated by reason of their viscid condition. They may be still better studied by receiving the blood from a prick of the finger at once into a solution of osmic acid (1 per cent.), or by placing a drop of blood on a slide and quickly washing it off with normal salt solution. In the latter case the plaques stick to the glass and are retained.

Fixed preparations. The study of fixed preparations of the blood is one of great interest, and has been placed among the well-recognized procedures mainly by the labors of Ehrlich and his pupils, though known before. The accuracy and the importance of the results, and the

pictorial beauty of the fixed and stained preparations, make Ehrlich's methods worthy of careful consideration.

If a droplet of blood be brought upon a cover-glass in the manner described for fresh preparations, then spread out into a thin layer by pressing the cover-glass upon a second, and the two glasses then slipped apart and allowed to become dry in air, a film of blood corpuscles is obtained in which scarcely any perceptible alteration of the shape or character of the corpuscles has occurred. It is advisable, according to Ehrlich, to hold the cover-glasses with forceps during their preparation, as the moisture about the finger seems to exercise some effect on the characters of the corpuscles. The thinness of the layer, and the consequent separation of the individual corpuscles and the rapid evaporation of fluid, prevent any degenerative changes which might mar the examination; the cover-glasses so prepared keep indefinitely with little deterioration. I have fixed and stained such cover-glass preparations after they had lain three years, and found the corpuscles of ordinary appearance and normal in their color reactions. For certain purposes the blood requires no further preparation, but may be examined or stained at once. Thus Gabritschewski demonstrates the presence of glycogen in certain leukocytes in such preparations by treating with a solution of iodide of potash 3, iodine 1, and water 100, to which has been added excess of gum arabic. If now one of these air-dried, but not fixed, preparations be stained with ordinary staining fluids (eosin, methyl-blue, etc., in water, weak alcohol, or glycerin) it will be found that the white corpuscles stain more or less well, while the red become paler and unstained, so that some further procedure is necessary to fix the hæmoglobin in the red corpuscles. This may be accomplished to a certain extent by using a solution of eosin in carbol-glycerin (5 per cent.), the carbolic acid acting as a fixing agent, but it is effected much better by first fixing the preparation thoroughly and then staining it.

Solutions of osmic acid, bichloride of mercury, picric or chromic acid, and Flemming's solution have all been used as fixing agents, and one or the other may still be needed for definite purposes, but the value of heat has been so clearly demonstrated by Ehrlich that the fluids named are seldom used. A mixture of equal parts of absolute alcohol and ether, however, is admirable for fixing the blood, and in ordinary clinical work possesses the great advantage of rapidity of action and ease of application, a dried cover-glass being simply placed in the mixture for a few minutes to half an hour.

Heat may be used in the way Koch advises for sputum, but the results are far better when the cover-glass is placed in an oven or on a

heated brass plate at a temperature of 100° to 120° C., for an hour to twelve hours. The use of a brass plate is simpler than that of the oven. A plate about a foot and a half long, a few inches wide, and a quarter inch thick is placed horizontally on a tripod and heated with a Bunsen flame at one end. After some time drops of water are dropped upon it, and the distance at which these do not assume the spheroidal state, but simply steam and disappear, is noted. This line roughly indicates the point of requisite temperature, and the cover-glasses are placed along this line or a little further distant from the flame. Preparations so fixed receive the stains in every portion and in definite manner.

The red corpuscles are evenly and distinctly colored, presenting a homogeneous protoplasm, as in the fresh condition. Sometimes, however, when treated in a special manner, as described by Foa,[1] a granular appearance is noted. Again, Ehrlich has found a fine reticular appearance in the stroma after staining with his eosin-hæmatoxylin mixture.[2]

Still another peculiarity of the red corpuscles, developed by staining, has been described—first by Celli and Guerneri, later by Ehrlich and by Gabritschewski. As we have seen before, the red corpuscle does not receive stains when in the unfixed condition; that is, it is achromatophile; under the influence of heat or reagents it suffers certain changes which render it chromatophile, but in such manner that the tint assumed is a simple one and like that of the stain employed. In certain cases, however, some of the red corpuscles do not behave in this normal manner, but instead are stained in tints varying from those of the substances used. Thus instead of becoming blue with methyl-blue, a greenish or purplish color is presented. This condition was found by Ehrlich to affect mainly the large macrocytes of anæmic blood, and was ascribed by him to degenerative changes. Gabritschewski, however, found the younger corpuscles of frog's blood to present this peculiarity, and concludes that it results from regenerative or formative processes rather than the reverse. My own attempts at repeating the work of either observer have been signal failures, and I am inclined to think too much stress has been laid upon peculiarities which have not yet been shown to be definite.

The nucleated corpuscles spoken of before are easily and beautifully stained with Ehrlich's eosin-hæmatoxylin, and may be seen to be of varying sizes, the normoblasts, the microblasts, and megaloblasts—names

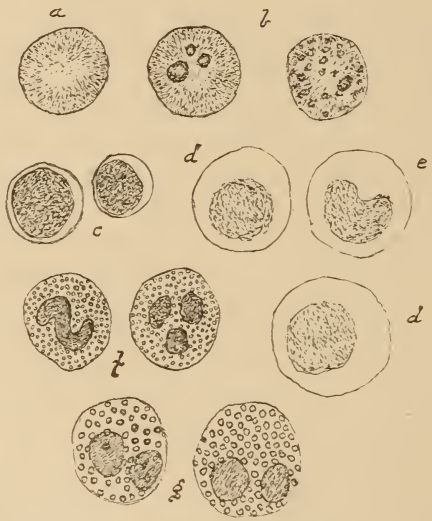
[1] Spread the blood rapidly on a cover-glass; dry in air; draw through Bunsen flame three times; stain with concentrated alcoholic solution of methyl-blue; wash; dry; clear with clove oil, and mount in xylol-balsam.

[2] Water, glycerin, alcohol, each 100 c.c.; hæmatoxylin, 1–2 gm.; alum to sat.; eosin, 1 gm.

indicating that they are equal to, smaller, or larger than red corpuscles. Karyokinetic figures may sometimes be demonstrated by the method given below for white corpuscles.

The staining of the blood plaques is so similar to that of the erythrocytes, or red corpuscles, that nothing further need be said concerning them, and we may pass on at once to consider the properties of the white corpuscles in detail.

Leukocytes have long been classified in four varieties: (1) Lymphocytes, generally smaller than the red corpuscles, with a nucleus almost entirely filling the cell and staining intensely; (2) large mononuclear elements, often several times the red corpuscles in size, with a somewhat



a. Red corpuscle, normal. *b.* Red corpuscles, showing highly refractive bodies. *c.* Lymphocytes. *d.* Large mononuclear leukocytes. *e.* Transitional leukocyte. *f.* Polynuclear leukocyte filled with neutrophile granules. *g.* Eosinophile leukocytes. The large mononuclear and transitional forms are usually larger proportionately than those here represented.

large and oval nucleus, which does not stain so intensely as the first; (3) transitional forms, identical with the last, and probably an intermediate stage between those and (4) the polynuclear or polymorphous, which are smaller but still of greater size than the red corpuscles, and in which the deeply staining nucleus has a highly irregular shape, not rarely S-shaped, and frequently divided into several smaller nuclei or fragments. The lymphocytes no doubt originate in the lymphatic glands and constitute about 20 to 30 per cent. of all the leukocytes. The origin of the second or third was thought by Virchow to be the

lymph glands also, but of the constancy of this there is some doubt; they constitute together 6 to 8 per cent. of all forms. The polymorphous possibly take origin in the spleen and constitute 70 to 80 per cent. of all forms.

Another classification has been established by Ehrlich, based upon the color reactions of the granules found in the leukocytes; but as an understanding of their differences requires a knowledge of the properties of certain dyes, a word or two is necessary upon this point. Coloring matters, especially the aniline dyes, may be divided into the acid and the basic, the coloring property residing in the former, in the acid of the compound, as the picric acid of picrate of ammonium; in the latter in the base. It is needless to enter upon details, but it has been found that among the former must be reckoned eosin, aurantia, picrate of ammonium and orange; among the latter, methyl-blue, methyl-green, dahlia, and others. Combinations of certain of the acids with basic stains produce neutral mixtures, which give rise to combination colors. Now, according to the behavior toward these different colors, or combinations of them, Ehrlich has described for human blood five forms of granules, two of which may, for the present, be disregarded, leaving three principal ones—the acid, the basic, and the neutrophilic. The acid and neutrophilic granules are found in normal blood, the basic occurs only in the blood of disease.

The *a* granulations, those which have a special affinity for acid stains, especially eosin, and are therefore called *eosinophile*, are found in leukocytes of various sizes and characters, though usually seen in large cells having one or two large nuclei. The granules themselves are seen in fresh blood as quite coarse or fine rounded bodies, suggestive at once of droplets of fat by their yellow color and their high refractive power. Their color has suggested to Hayem and others that they contain hæmoglobin. That they are not fat is abundantly proved by the fact that the preparations may be kept long periods of time in absolute alcohol or ether without destruction of the granules, that they do not stain black with osmic acid, and that fat does not stain as these granules do. That they are not hæmoglobin-containing is also seen in their behavior to stains. Ehrlich suggests a peculiar albuminous nature, but with no certain justification.

These granules have the greatest affinity for eosin, though other acid stains are well received. Good results may be obtained by staining the air-dried and heated cover-glass in a saturated solution of eosin in glycerin for twelve hours or more; but for clinical work I find the following method most satisfactory: the air-dried cover-glass is placed in a

mixture of absolute alcohol and ether five minutes, then stained with a 1 per cent. solution of eosin in 60 per cent. alcohol, to which has been added an equal quantity of water at the time of staining. The cover-glass is left in this solution a minute or two, until it assumes a decided pink color; washed in water; counter-stained in Delafield's hæmatoxylin about one minute and again washed, then dried and mounted. The eosinophile granules are dark red, the red corpuscles lighter, the nuclei of the white corpuscles deeply colored by the hæmatoxylin. The prettiest effects are obtained from glycerin solutions, and for differential staining a mixture[1] of indulin, aurantia, and eosin may be used. With this stain the nuclei are purple or black, the red corpuscles orange, the eosinophile granules red.

The claim of Ehrlich, that the presence of these eosinophilous leukocytes in large numbers is diagnostic of leukæmia, has not been sustained, and he has himself abandoned the view. The specimen I show you is from a case of asthma, and, like another from the same disease which I have here, shows distinct increase of eosinophiles, without leukocytosis of marked degree. The counting of the number of eosinophiles may be accomplished roughly by moving the slide from field to field and counting all the leukocytes in each until a large number have been put down. This is a tedious and crude method; and a better has lately been proposed by Zappert, which, however, I have not yet used. He employs with the Thoma-Zeiss instrument a diluting fluid[2] containing eosin, and counts these, as other corpuscles, on the slide. The solution stains the eosinophile cells dark red, the neutrophilic and red corpuscles light pink, after a few minutes.

The neutrophilic or ε-granulations are the most abundant granules of the normal blood, and, when properly stained, are seen as fine bodies closely packed and filling the whole of the polymorphous leukocyte. The latter always contains such granules, and in healthy blood none of the other white corpuscles presents such. In leukæmia, however, the same

1 Sat. sol. aurantia in glycerin . . . . . 1 part.
Add glycerin . . . . . 1 or 2 parts.
Eosin and indulin in excess.
Mix by prolonged agitation.—(Schwarze.)
Or,
Indulin, Aurantin, Eosin } each . . . . . 2 parts.
Glycerin . . . . . 30 (Huber.)

2 Osmic acid sol. (1 per cent.) . . . . . 2 parts.
Glycerin and water each 25, Aqueous sol. of eosin (1 per cent.) 100 } . . . . . 1 part.

granules are seen in the large mononuclear and transitional forms, and the same kind of corpuscle is normally found in the bone-marrow, whence Ehrlich readily arrived at the conclusion that the cells are myelogenic and gave them the name of "myelocytes." They occur only, as was said, in leukæmia, but are not invariably present in this disease. Various mixtures have been proposed as neutrophilic stains. Ehrlich first used a mixture[1] of methyl-blue or green with acid fuchsin; and with this the neutrophile granules are of purplish color, the eosinophile dark red, the nuclei blue or green. Later the same author proposed solutions[2] of acid fuchsin, methyl-green, and orange G, with which the neutrophile and eosinophile granules have the same appearance as with the other, but the red corpuscles have an orange tint.

The basophilic or γ-granulations resemble bacteria in their reaction with aniline dyes, taking such basic stains as methyl-blue and dahlia by preference. They are sometimes abundant in leukæmic blood, and are present in various connective tissues, where their apparent relation to nutritive processes caused Ehrlich to give them the name "mastzellen." They are the same cells as those which Waldeyer called "plasma-cells," or at least some of the latter are mastzellen. Their significance has not yet been determined. Ehrlich suggested dahlia[3] as a stain, but methyl-blue[4] is also efficient, though my experience has been mainly with the former.

[1] Sat. aq. sol. of acid fuchsin . . . . . 5 parts.
Sat. aq. sol. methyl-blue (or green) . . . . . 1 part.
Add the second to the first slowly, stirring vigorously, then add
Distilled water . . . . . 5 parts.
After a few days filter.
Stain 2 to 5 min.; wash hastily in water.

[2] Sat. aq. sol. of orange G. . . . . 125 c.c.
Sat. sol. (in 20 per cent. alcohol) of acid fuchsin . . . . 125 c.c.
Mix, and add gradually, shaking constantly,
Sat. aq. sol. of methyl-green . . . . . 125 c.c.
And finally
Absolute alcohol . . . . . 75 c.c.
Let the solution stand several weeks, and take out the required quantities with a pipette. *Do not filter.* Biondi has modified the formula slightly, and his mixture of dyes, ready for solution, is kept by large dealers. One part of the mixture with six of water and one of absolute alcohol makes the proper solution.

[3] Dist. water . . . . . 10.0 c.c.
Sat. sol. of dahlia in absolute alcohol . . . . . 50 c.c.
Glacial acetic acid . . . . . 10–12.5 c.c. (Ehrlich.)
Müller uses this solution, staining specimens (which were first heated and then placed in absolute alcohol several days) for several hours, after which they are washed in water and a considerable time in alcohol.

[4] Sat. aq. sol. methyl-blue . . . . . 40 c.c.
Sol. of eosin (½ per cent.) in 70 per cent. alcohol . . . . 20 c.c.
Distilled water . . . . . 40 c.c.
The air-dried specimens are put in absolute alcohol at least five minutes, then in the stain, where they are kept at a temperature of 37° C. for 3 to 6 hours. Wash in water. The eosinophile and basophile granules are prominently stained. (Canon.)

I need not multiply the methods of study by enumerating the many which have been proposed, but will rest content with having given the best, and those which, for the most part, I have had occasion to employ. The subject of the "specific granulations" of Ehrlich is sufficient to have engaged the amount of time I have devoted to the whole question of blood examination, but it was not the intention of your committee, or my desire, that I should enter upon the discussion of the nature and origin of these structures, or upon the other interesting questions arising from these studies. Whatever of a practical nature we may admit or deny to these studies, all will concede the immense debt of gratitude our science owes to Ehrlich for having by this means at least aroused a renewal of interest in the important field of hæmatology.

One more point and I have done. Müller has recently studied the question of karyomitosis in white corpuscles, a subject to which attention was first directed by Flemming. Müller fixes the blood by heat and saturated solution of picric acid, placing the heated cover-glass in the latter for twenty-four hours. The preparation is then washed in running water for several hours and stained in much-diluted Delafield's hæmatoxylin for twenty-four hours; or the cover-glass may be taken from the picric acid and at once put in borax or ammonium carmine. Müller found in leukæmic blood certain large cells resembling Cornil's marrow cells, which presented distinct karyokinetic figures, and which he regards as characteristic of leukæmia. The observation requires confirmation. *December* 22, 1892.

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28. *Pericardial effusion and mitral stenosis.*

Exhibited by Dr. H. M. Fisher.

H. T., aged 21 years, was admitted to the male medical ward of the Episcopal Hospital, November 4, 1892. For the following clinical record I am indebted to my resident physician, Dr. S. P. Eagleton.

Patient's friends stated that he had never been strong, and for years had been very much fatigued by slight exertion. Upon admission face pale, lips cyanosed, extremities cold, very feeble pulse; much dyspnœa. His chest was of the pronounced "chicken-breast" type, showing marked narrowing at its anterior segment. Præcordial dulness extended from a point one and a half inches to the right of the sternum to a point about one inch to the left of the left nipple; apex-beat in the seventh interspace. Auscultation showed rapid and tumultuous action of the

heart, and there was a loud systolic apex murmur. A presystolic thrill was also felt, with its point of greatest intensity a little to the left of the xiphoid cartilage. Both lungs posteriorly showed marked impairment of resonance at their lower thirds.

Treatment consisted in the administration of digitalis in large doses—fifteen minims of the tincture every two hours at first and strychnia at the same time. The patient was relieved a little at first, but on the night of the 10th he became suddenly worse and cyanotic to an extreme degree. He died at 9 o'clock on the morning of the 11th of November, one week after his admission.

Autopsy twenty-four hours after death. Pericardial sac distended with fluid, and, with the heart, occupied the whole anterior portion of the thoracic cavity. Parietal surface of pericardium thickened and adherent to chest-wall. Fifty fluidounces of serum were removed from its cavity.

Heart. Right auricle holds an entire fist. Right ventricle much dilated, and contained numerous currant-jelly clots. Tricuspid orifice dilated. Left auricle also much dilated, walls thickened and atheromatous plates on inner surface. Mitral admits only the point of the little finger, and is "button-hole" in shape. *Lungs.* Left adherent at lower border. Twelve ounces of serum in left pleural cavity and sixteen in right. An area of infarction in posterior portion of middle lobe of right lung. *Kidneys.* Surface uneven; capsule non-adherent; cortex diminished. *Liver.* Purplish discoloration, "cyanotic induration." In the upper anterior surface of right lobe white nodule of the size of a large pin's head and apparently an old infarction.

Dr. Stengel called attention to the extreme rarity of infarcts in the liver, and questioned, therefore, the nature of the formation alluded to by the speaker. At any rate the statement could not be admitted unless supported by decided proof. Dr. Stengel had himself seen, with Prof. Allen J. Smith, a fresh infarction about which they entertained no doubt, but which had unfortunately been thrown away by the servant of the laboratory on the day of the meeting at which Dr. Smith proposed presenting it to the Society.

Dr. Fisher, in reply, stated that he had used the expression infarct in a somewhat routine manner, without actual proofs.

January 26, 1893.

29. *Thrombosis of the heart and pulmonary artery in pneumonia.*

Exhibited by Dr. J. M. Miller.

The patient was admitted to the Episcopal Hospital pulseless and cyanotic. He had laid in a barn for seven days. The breathing in the right side was suppressed; on the left it was bronchial.

At the autopsy the lungs were much congested, especially posteriorly, while anteriorly there was some emphysema. There were spots of catarrhal pneumonia on both sides. The heart contained in both chambers dense white clots which extended as far as the carotid vessels in the greater circulation, and in the lesser as far as the minutest ramifications of the pulmonary artery. The extensive character of the thrombosis seemed of sufficient interest to claim the attention of the Society.

January 26, 1893.

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30. *Heart and aorta showing calcareous plates and atheromatous ulcers.*

Exhibited by Dr. Lawrence Wolff.

August H., aged 46, white, on admission gave a history of having been troubled with attacks of dyspnœa for some years. Was very thin and emaciated. Had great trouble in breathing, and could not rest in a recumbent position. Was very icteric. Had all the symptoms of an aneurism of the arch. Radial pulse on left side almost entirely absent. Bulging of the supra-clavicular region on right side. Pulsating jugulars. Heart sounds so irregular and mixed up that the murmurs could not be located, although they were heard.

Post-mortem examination showed the lungs to be normal, with the exception of being slightly œdematous. Pericardium empty. Heart showed left ventricle hypertrophied, with dilatation. Right ventricle and auricle greatly dilatated. Slight dilatation of arch of aorta. All the valves and the aorta atheromatous. Calcareous plates found in arch, almost entirely occluding the left subclavian artery and left innominate.

Liver cirrhotic. Other organs normal. *January* 26, 1893.

31. *Ulcerative endocarditis.*

Exhibited by Dr. LAWRENCE WOLFF.

George P., aged 41, blacksmith by trade. Had history of having had attacks of typhoid fever, scarlet fever, and rheumatism. Had always been a hard worker when in health. On admission to hospital was very weak, cyanotic, and in a condition of general anasarca. Abdomen largely distended with ascitic fluid. Mitral murmur heard. Was icteric. Died from œdema of the lungs.

Post-mortem examination showed the chest filled with serum, having the appearance of being bile stained. Lungs œdematous; right lung showing old pleuritic adhesions. Pericardium was filled with serum. The heart was hypertrophied. The aortic valve showed warty vegetations, with ulcerated button-holes. Mitral valve almost entirely absent, with warty growths around the edges. Right side of heart normal.

Liver showed a peculiar pigmentation, brown and red, with the general appearance of a nutmeg liver, and was considerably enlarged. Spleen was enlarged. Kidneys were of the large white variety, with a small cyst in the cortex of the left. *January* 26, 1893.

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32. *Aneurism of the arch of the aorta.*

Exhibited by Dr. LAWRENCE WOLFF.

John Q., aged 30, driver by occupation. Chief symptom while in hospital was very marked dyspnœa. Heart sounds very feeble, but no murmurs or friction sounds heard. Pulse fairly strong on right side, but entirely absent on left. Patient suffered from attacks of cyanosis, and was hardly able to draw his breath.

Post-mortem examination showed an immense dilatation of pericardial sac, with about a litre of fluid in the sac. Pleural sacs free of fluid. Fibrinous pericarditis nicely shown. Right ventricle enlarged and marked left-sided hypertrophy. Valves healthy. Aneurism of transverse arch about the size of an apple. Trachea had superficial ulcerations, but no rupture. The beginning of the left subclavian showed slight dilatation, into which the opening of the artery projected, somewhat resembling a nipple projecting into a pouch.

Liver increased in size. Lungs, stomach, and intestines normal.

Kidneys showed on right side enlargement, with grayish coloring. Cysts found which contained cheesy matter resembling curdled milk. Left kidney very large, but apparently normal; functional hypertrophy. *January* 26, 1893.

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33. *Hemorrhagic pericarditis.*

Exhibited by Dr. Lawrence Wolff.

Jacob F., aged 67, weaver. Very weak on admission to hospital. Had a rapid, running pulse, apex beat diffused and displaced to left. Impaired heart sounds. No murmurs or friction sounds.

Post-mortem examination showed dilated pericardial sac, with parietal layer adherent to visceral layer at upper part by shaggy bands of lymph.

Heart and lungs normal. Liver enlarged, with abundance of connective tissue. *January* 26, 1893.

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34. *Enlarged heart with valvulitis.*

Exhibited by Dr. S. Solis-Cohen.

The specimen presented was removed from the body of an old man who died at the Philadelphia Hospital very soon after admission to the wards. There had been most extreme dyspnœa and widespread œdema. The subcutaneous infiltration did not have the characteristics of a pure œdema, but rather those of a mucin-like deposit. The tissue could be pinched up, and seemed firmer and more substantial—perhaps gelatinoid would be a better term—than dropsical tissue. Puncture of the greatly swollen scrotum, made to determine this point, gave exit to but a few drops of fluid, and at the autopsy gelatinous material was found in the subcutaneous tissue.

At *autopsy* an interesting condition of the heart was found. Its weight was twenty-five ounces. The aortic leaflets were uniformly thickened and rigidly projected into the lumen of the aorta, causing stenosis, and at the same time insufficiency. The mitral valves presented nodular swellings on the auricular and ventricular surfaces. The other valves were healthy.

There was also evidence of an old appendicitis healed by fibrous adhesions.

The history of the patient gave no definite information as to the cause of the endocarditis. The man gave a history of influenza, but had never had rheumatism.

Dr. Stengel referred to the great obscurity in the etiology of many cases of valvular disease. Of course, it is well known that a majority of all cases, perhaps a large majority, take origin in acute rheumatism, and especially is such the case when the patient is a young man and the mitral valve is the one affected. In the present case, however, the greater age of the patient and the special implication of the aortic valve leads our thoughts to other factors of causation. Alcoholism, syphilis, gout, and nephritis all play an important part, as does also prolonged muscular strain, such as is necessitated by the hard work of many of our laboring class. This point had been called to the speaker's attention very forcibly within the last few days by a case which had fallen under his observation. This case was a man under 30 years who suffered with extreme mitral regurgitation. In him there was no history of rheumatism, infectious fevers, syphilis, alcoholism, or gout. His history was remarkably clear, and his habits had been beyond reproach, so that there can be no question of his statements. But the element of muscular strain was decided in his case. He began work on his father's farm at a ridiculously early age, and continued until old enough to perform more difficult labor. During late years he had been employed loading and unloading stone and gravel into a wagon, and then into a boat. The strain was great even for one physically as well made as he, and at night he was always completely exhausted. Now here was only one etiological factor, and curiously enough it was the mitral valve which suffered change. One might be almost led to question the accuracy of the deductions from this fact, but the most careful search failed to discover antecedent rheumatism or other causes of previous endocarditis. The influence of extreme muscular strain and high arterial pressure must naturally be exerted with great force upon the valves. This was proved experimentally by Roy and Adami. These observers placed ligatures about the aorta of dogs, and made constrictions for given lengths of time. The effect was found to be a rapid change in the valves, their thickness increasing and the subendothelial tissues often being infiltrated with blood. The frequency of chronic endocarditis in soldiers, miners, blacksmiths, and others whose work is laborious finds ready explanation in the increased arterial pressure induced by muscular strain; and, as Albutt has shown even young

men show advanced sclerotic changes when their work has been of the nature described.

Dr. Eshner was disposed to lay great stress upon the importance of the infectious fevers. The whole group of fevers must be taken into consideration, since any one may occasionally lead to valvular disease. In one of his own cases puerperal infection alone seemed admissible as the cause. We are very much in the dark about rheumatism and its cause, but there seems much reason to believe in its toxic nature. The occurrence of endocarditis, at any rate, is well explained by such an assumption, and in this point acute rheumatism is to be classed with the infectious fevers, though of course its influence is more constant and widespread than that of any of the latter.

Dr. Meigs referred to the case of an elderly lady who had been under his observation for over thirteen years, during which time she had had various ailments, but no sign of heart afiection such as we are accustomed to recognize. Shortly before death, however, the evidence of valvular disease became unmistakable, and at death the condition was found in an advanced degree. He therefore felt that very many cases of valvular disease have an insidious nature, and do not begin as acute processes. Certainly such was the case in the lady referred to.

Dr. Ball, in the course of his examination of 450 criminals, most of them apparently healthy, had found 25 with organic heart murmurs. In one-half of them a distinct history of rheumatism was obtained. In 20 there was no history of inconvenience from the lesion.

Dr. Cohen in closing remarked that it is important to distinguish between the real inception of the disease and the onset of the symptoms. The latter are developed only when some strain or intercurrent disease has taxed the cardiac power and led to disturbance of circulation. The influence of infections cannot be overlooked in discussing the etiology of endocarditis in any of its forms. He was convinced, moreover, that apparently trivial causes, such as tonsillitis, are oftener the cause of endocardial mischief than we might be disposed to admit. Excess of uric acid in the blood is also a cause to which he would look with care. It is not necessary, however, that there should be an acute beginning. Many cases of valvular and muscular disease of the heart are doubtless chronic from the first; that is to say, they are of gradual and insidious development, due to causes working more or less continuously over a long period of time. *February* 9, 1893.

35. *Renal thrombosis, with pulmonary emphysema, in an infant six weeks old.*

Read by Dr. ARTHUR V. MEIGS.

An infant, which appeared to be about 4 weeks old, was admitted to the Sheltering Arms, January 15, 1889. The child was a female and was well nourished, and presented no evidence of disease except slight inflammation of the eyes, which disappeared a few days after her admission to the institution. She was given cream, one ounce; milk, half an ounce; lime-water, one ounce; and sugar-water, one and a half ounces, with a teaspoonful of Mellin's Food. The sugar-water was made of eighteen drachms of milk-sugar to one pint of water. For a time the infant appeared to thrive, but upon January 26th she became very fretful. On the 27th aphthous sores appeared in the mouth; she vomited, and the movements were yellow. On the 28th the features became pinched and the nostrils dark; the breathing was often irregular, and she had a distaste for food. The mouth was washed with a solution of chlorate of potash, and she took thirty drops of brandy every three hours. January 29th, the mouth was better, but the general condition of the infant was worse, and she appeared to suffer great pain, especially on being moved. Toward evening she died. The nurse said that at times she had passed large quantities of urine and again very little.

Post-mortem examination was made eighteen hours after death. The body was well nourished, there being a good deal of subcutaneous fat on the chest, abdomen, and limbs. (The contrary is common in foundlings, the absence of adipose tissue being usually marked.) The brain, medulla oblongata, and spinal cord appeared to be normal. The lungs looked healthy, except that upon the anterior surface, at the base of the upper lobe of one of them (it was not noted whether the right or left) there was upon the surface a bleb about one-half to three-quarters of an inch long, which contained air and seemed to be subpleural. The heart, thymus gland, liver, spleen, uterus, and ovaries appeared normal. It is worthy of note that at this time of life (six weeks) the cervix uteri is wider than the fundus. The intestines and mesenteric glands were natural in appearance, and the bile-duct was patulous. The pancreas appeared to be healthy; the hypogastric arteries were still open. The stomach was small and contracted, and contained some dark and slimy material which resembled altered blood. Microscopical

examination of this material, however, showed it to be composed of dark pigment without recognizable character, oil drops, quantities of mucous, a few starch granules, and epithelial cells. No blood corpuscles were found in it. The kidneys had upon their posterior surface quite a thick layer of fat. On section there appeared one spot which looked as if hemorrhage into it had taken place, this occupying about one-half of one of the cones. Several of the cones toward their orifices were yellowish in color and appeared to contain some saline material. In many places at the junction of the medullary and cortical portions there were to be seen on section yellow spots. It was found that these were clots in the vessels, of which strings a quarter of an inch long in some instances could be pulled out. Sections were prepared for microscopical examination of the tongue, epiglottis, and trachea; of the lung and of the aorta; of the œsophagus, stomach, and duodenum; of the pancreas, ileum, and mesentery, including glands; of the liver, spleen, and of the kidneys from two different positions. Many of the sections presented no recognizable evidence of disease. The section of the lung was cut so as to include the bleb upon the surface. Upon its outer free side the wall was made up of a thin fibrous and non-vascular substance. Next to the lung the wall of the sac was fibrous, but somewhat thicker and quite rich in nuclei. There appeared to be no communication with the lung. In the section the bleb was half an inch across in its largest diameter, although it must have contracted somewhat in process of preparation. The general impression conveyed by the examination of the lung is that the air-spaces are larger than usual at this period of life, and possibly the alveolar walls less vascular and more fibrous than natural. It would not be more than might naturally be expected, as one large emphysematous bleb did exist, that the rest of the lung should show evidence at an early stage of what had in the one instance progressed so far. The walls of the arteries appear thicker and more fibrous than usual. Thus the appearances seem to be parallel with the common pulmonary degenerative changes of later life. The spleen had shreds of tissue upon it that looked like the remains of adhesions. Microscopical examination of the kidneys shows that the large thrombi were in the veins; they were very numerous, and the veins were distended to three or four times their usual size, as seen in microscopical preparations. In some positions blood had escaped into the tissue. There are many casts in the tubules in the medullary portions. Microscopic examination showed the presence of saline material, probably urate of ammonia in the straight tubules. The capsule is of decidedly varying thickness at different places, and masses of peri-

renal fat are included in the sections, these being as usual adherent to the capsule. As places beneath the capsule there are areas in which cell infiltration of slight degree seems to have occurred—the appearance seeming to be parallel with the contraction which is so common at later periods of life. It would seem that there was moderate increase of the fibrous material in the bloodvessels. This could be distinguished in the pancreatic artery and vein, and in an artery and vein included in a section of the mesentery. This was, however, of the outer coats of the vessels and perivascular connective tissue more than of the intestine, as is so common in older persons.

Whether the death of the infant was caused by the thrombosis of the renal veins or not it is impossible to say. It was interesting, however, to me to see renal thrombosis at so early a period of life as six weeks. The case has a value, too, as an exemplification that degenerative pulmonary changes, which are so common in older persons, may be present in so young an infant, and one that appeared to be in a good state of nutrition. The pulmonary changes are certainly to be considered as of the nature of, if they are not naturally, emphysematous.

March 23, 1893.

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36. *Malignant endocarditis.*

Exhibited by Dr. W. E. Hughes.

The patient gave a history of rheumatism for twenty-four years, chills and fever, and gonorrhœa. Six weeks before he came under observation he grew dyspnœic, and could not work. Apex beat about two inches outside of nipple line. Impulse diffuse. Marked systolic murmur, marked diastolic aortic sound, and occasionally a systolic aortic sound. Later tricuspid regurgitation developed.

Autopsy. Heart large, both ventricles much dilated. Mitral valves bound down, aortic valves roughened and incompetent, and on one edge there was a warty growth and a perforation of the valve. Some aortic atheroma.

Dr. Stengel remarked that gonorrhœa has lately been found to occasion certain cardiac affections, among which is malignant endocarditis. Very recently acute myocarditis has been added to this list.

May 11, 1893.

37. *Acute pericarditis.*

Exhibited by Dr. Henry W. Cattell.

B. C., aged 50 years. The specimen is shown simply as one of those typical cases where the visceral and the parietal layers of the pericardium are commencing to be glued together by means of a fibrinous exudate, giving the appearance of a cat's tongue when the layers are torn apart. The fibrinous deposit covered the entire heart. About 300 c. c. of fibro-purulent fluid was found in the pericardial sac. There was also an acute endocarditis of the mitral valve.

The pericarditis was apparently secondary to pneumonia of both of the lobes of the left lung. *May* 11, 1893.

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38. *Large dissecting aneurism commencing in the descending arch of the aorta and ending in the lower portion of the abdominal aorta.*

Exhibited by Dr. Henry W. Cattell.

T. F., aged 54 years, had a distinct history of syphilis and rheumatism. He was also accustomed to use large quantities of alcohol. The aneurism was not suspected during life. I am indebted to Dr. J. H. Girvin for the following clinical notes.

About four years ago the patient began to lose the power of his legs, with paræsthesia in the feet and a feeling of numbness of soles and some tingling, and subsequently gradually increasing loss of power, especially in right leg. He has had constriction about the legs and an ill-defined girdle sensation. He has not had incontinence of urine or feces, but some difficulty in micturition, and bowels have been moved without distinct sensation of the passage.

At the present time the right leg is quite helpless, although the feet are moved more freely than the upper leg. Skin reflexes gone. Knee-jerk absent, but distinctly present by reinforcement. Sensation acute, although localization not perfect. Left leg is much less affected than the right; can lift thigh and move foot quite freely. Knee-jerk also absent and not reinforceable. Skin reflexes same as right. Has no sharp shooting pains in legs. Elbow-jerk is present on both sides. Pupils contracted but respond to light slightly, and to accommodation. There were throbbing vessels, a mitral murmur, and a double aortic.

Post-mortem. The aorta is atheromatous throughout its entire extent. The vessels of the arch come off normally. In the descending portion of the arch are situated two abnormal openings; the first is transverse and ovoidal in character, and arises half an inch from the left subclavian artery. It is ten lines long by five lines broad. All the measurements were made after the specimen had been in about 70 per cent. alcohol.

Ten lines from this, further down, is another opening smaller and more nearly circular, six and one-half by four and one-half lines. These openings communicate with a large cavity the size of a small orange, not affecting the subclavian, though at its commencement there is a small pocket dilatation of the arch. The aneurism had then dissected its way down the thoracic and abdominal aorta, lying chiefly on the left side, the dilatation gradually diminishing until within several inches of the bifurcation; here are six distinct openings, the largest one being ten lines above the bifurcation, and is transversely ovoid, looking like a tubercular ulcer in the intestine; it is nine and one-half lines long by six and one-half lines across. Just above this are three smaller openings opposite bloodvessels, and about three-quarters of an inch above these two larger openings.

On the outside the wall of the aneurism measures twelve and one-eighth inches, and fourteen and three-fourths on the inside. The greatest outside circumference of the aorta and aneurism at the descending arch being six and seven-eighths inches, the aorta proper being but three and one-eighth inches for the internal circumference.

The greatest point of interest consists in the fact that some of the abdominal vessels, as the left renal, are given off at once from the wall of the dissecting aneurism. The mesenterics come off from the aorta directly. In other words, there is an opening, the former place of exit of the artery, from the aorta into the aneurismal sac, and then at a point opposite the vessel comes off, showing that the attachment of the artery from the aorta had been torn off and that still the blood was able to circulate to the parts being supplied by means of the blood entering into the aneurism from the openings in the descending portion of the arch of the aorta, and also by an opening left in the aorta from which the vessels had originally come off. The intercostals come off directly from the aorta and do not communicate with the aneurism, though in places the sac is beginning to involve their points of exit.

On making a transverse cut through the aorta and the aneurism the appearance is as if there was a double aorta surrounded by a single coat. There is a lining membrane which as far as the macroscopic appearance goes is similar to that of the aorta. The aneurism is in the

muscular coat, as stated above, the adventitia surrounding both the aorta and the aneurism. The wall between is thick and strong, there evidently being an increased amount of new connective tissue. The inside wall of the aneurism contains hundreds of scaly calcareous plates.

The heart is hypertrophied and the ventricles dilated. Weight estimated at eighteen ounces, not accurately determined, as the aorta is attached. The edges of the mitral valve are slightly thickened. The aortic valves are thickened, and the following condition exists: The right posterior leaflet is firmly attached to the right edge of the anterior leaflet by means of fibrous and calcareous nodules extending about three-eighths of an inch directly out into the lumen of the aorta and extending downward, where it narrows into the sinuses of Valsalva. This is the only point of calcification in the valves. The commencement of the aorta is less atheromatous than the other portions. There is fatty infiltration and degeneration of the heart muscles. The pulmonary valves are normal except as to size, the artery being wider than normal, four and one-half inches in circumference at the attachment of the valves. The right anterior leaflet at the top is unusually large, and measures one and five-eighths inches and is five-eighths of an inch in depth.

It need hardly be stated that the process must have been a very gradual one, and to have continued for a long time.

The other pathological diagnoses were chronic parenchymatous nephritis, cirrhosis of the liver, and fat necrosis.

Dr. Stengel remarked that this case presented many interesting features from a clinical as well as pathological point of view. The clinical history given is sufficient to indicate the existence of a spinal lesion probably in some way connected with the specific history, and the patient was treated, with this idea in mind, by mercurial inunctions. Very curiously, after the second application the gums became affected and the treatment was discontinued. After a time, however, it was repeated, and again the patient became affected. At this time, however, instead of improving, the patient's condition became steadily worse, fever was developed, delirium, and finally coma set in, and the patient died without manifesting any symptoms which would explain the curious conditions.

The aneurism exhibited is certainly a most interesting specimen, and it is really a remarkable thing that the man's history had given no indication of the occurrence of the first tear. Pathologically this specimen well illustrates what Peacock and others who have studied dissecting

aneurisms claim, viz., that the dissection follows in the planes of the muscular layer; and still more strikingly do we see in the present case the new-formed lining of the aneurismal sac with endothelium. A careful inspection at the point of perforation shows a continuous endothelial lining, and the secondary atheromatous changes in the aneurism are in all respects similar to those in the aorta itself. *May* 11, 1893.

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39. *Malignant endocarditis.*

Exhibited by Dr. W. E. Hughes.

These specimens were removed from a man, of whose clinical history I can give but a meagre outline. He was a negro, 24 years of age. For over three months he had been complaining of a cough and of failing flesh and strength. Of late he had epigastric pain and pronounced dyspnœa, and finally syncopal attacks. Death was sudden.

The heart, you will see, is hypertrophied, but on the right side dilatation dominates over hypertrophy. Both sides were filled with chicken-fat clots. The aortic valves are the seat of marked vegetative and ulcerative lesions of malignant endocarditis. The ulceration has completely perforated the septum and extended to the tricuspid leaflets, which are likewise covered by vegetations.

Both pleural cavities contained a small quantity of clear serum, and a few tubercles were found near the apices of the lungs. There were anæmic infarcts in the kidneys, which in general were deeply congested.

The stomach was entirely normal, but the duodenum was much congested. The gall-bladder was greatly thickened, its walls presenting, on cross section, a peculiar colloid appearance, which excited some interest, but has now almost completely disappeared. It was this condition of the gall-bladder to which I desired to call your attention, but its disappearance proves it to have been an œdematous state of the tissues. The cystic duct was occluded, the hepatic patulous. The liver was of normal appearance.

The man had not had jaundice. There was no rheumatic history or any other condition to which the cardiac lesion could be attributed.

June 8, 1893.

40. *Double aneurism of the aorta, with rupture externally.*

Exhibited by Dr. ALFRED HAND, JR.

M. K., male, aged 48 years, married, white, born in America, a bricklayer, was admitted October 25, 1892. His history was that three years before he had had a right-sided empyema, which was tapped. A rib was resected and a drainage-tube inserted until the discharge ceased and the side healed. After this, he was in good health until about six months ago, when he began to be troubled with shortness of breath and painful deglutition. In a short time he noticed a small tumor close to the right border of the sternum. This steadily increased in size, and the difficulty in breathing and swallowing became greater. The appearance and growth of the tumor were marked by no special pain over it. He denied having had syphilis.

On admission he was found to have a pulsating tumor about the size of a hen's egg, over the third and fourth right costal cartilages, spreading also over the ends of the ribs. There were no heart murmurs, nor any thrill nor bruit over the tumor. The radial pulses were equal and the pupils regular. Resonance was impaired over the right lung, and the respiratory sounds were feeble, with prolonged expiration. These phenomena, with his former history, raised the question of an *empyema necessitatis*, but owing to the expansile pulsation of the tumor, the pain on swallowing, and the wheezing breathing, the diagnosis was made of aneurism of the ascending arch of the aorta.

For about five months the man's condition remained practically the same, the tumor growing a little larger. The patient was very restless, and could sleep scarcely more than two hours in the twenty-four. At times the tumor would recede somewhat into the chest, and he would then imagine himself better, but the difficulty in breathing and swallowing would be greater.

He left the hospital for two months in the spring of 1893, but came back in June, with the tumor projecting three inches from the chest-wall and extending over an area six by four inches. The overlying skin had ulcerated, and from the surface blood shortly began to ooze. About a week later the rupture came, the stream of blood being at first about a quarter of an inch in diameter and of sufficient force to strike horizontally the wall four feet away. At least twelve minutes elapsed from the first spurt until life was extinct.

The post-mortem examination disclosed an immense aneurism of the

ascending arch which had perforated the chest-wall, with erosion of the third and fourth ribs. At its exit from the chest it was constricted like an hour-glass, so that there were two large tumors, one on the inner side and the other on the outer side of the anterior chest-wall. A finger could be introduced through the point of rupture into the outer sac, and a soft clot and the perforation in the chest-wall could be felt. The inner sac could be explored by passing the finger up through the left ventricle into the aorta, which was lined with calcareous plates in its first part. The inner tumor also contained a large, soft clot. On the upper posterior part of the transverse arch was a sacculated aneurism about two inches in extent and an inch deep, containing a large lamellated clot. This aneurism was probably the one which gave rise to the difficulty in breathing and swallowing. The heart was normal, no valvular lesion existing. The left lung was normal, but the right was largely solidified from compression. *June* 22, 1893.

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41. *Aneurism of the basilar artery and anomalous circle of Willis.*

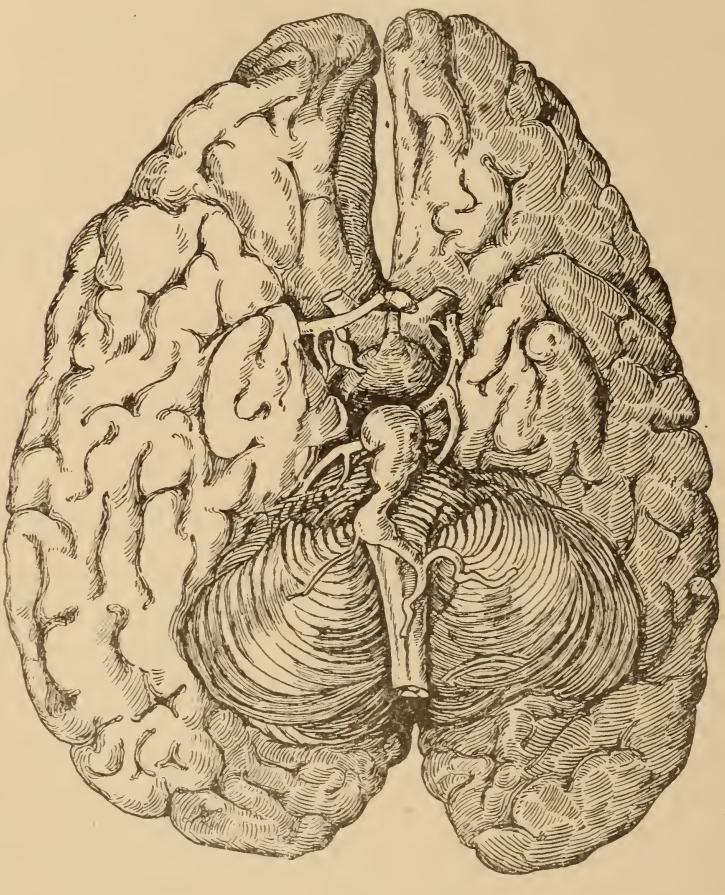
Exhibited by Dr. Henry W. Cattell.

I am indebted to Dr. George K. Edwards for the following clinical notes:

Mrs. A. R., 60 years of age, white, has shown signs of paralysis for some time, but has been able to be about. While sitting in a rocking-chair she attempted to rise, but fell back, and in falling the left arm was fractured above the wrist. The following day the patient seemed to be in a dazed condition, but could answer questions intelligently. The urine constantly dribbled away, but on catheterization it was found to be incontinence of retention, twenty-five ounces being drawn. Catheterization was employed twice a day and during the night, which nearly entirely relieved the dribbling. Complained of a great deal of pain in the arm. Three days later the patient became very drowsy and hard to arouse; was almost comatose. Pupils dilated and breathing rapid. Urinary analysis showed nothing suggesting uræmia.

Subsequently the patient became comatose, the breathing harsh and almost stertorous, and eyes persistently closed. All the symptoms pointed to compression of some part of the base of the brain, and in two or three days the patient died. There was a history of syphilitic infection dating to some fifteen years ago.

Post-mortem. The notes on the brain are alone given. Large amount of cerebro-spinal fluid. Arteries at the base of the brain very atheromatous. The calibre of the entire basilar artery is somewhat increased, widening until just before its bifurcation, where it expands into an aneurismal sac the size of a small hazelnut. The cavity is occupied



by fibrin, chicken-fat, and currant-jelly clots. The fibrinated portion is probably somewhat organized. There is an anomalous circle of Willis. The left vertebral artery is larger than normal, the basilar really being a continuation of it. The right vertebral crossing over to the left side, empties, as it were, into the left vertebral. The posterior cerebral arteries are of good size. The left posterior communicating is ex-

tremely short, being only about three-eighths of an inch long. A very small posterior communicating artery comes off from the right internal carotid, and instead of communicating directly with the posterior cerebral, runs several inches backward to the pons, back of the crus, and unites with a branch from the posterior cerebral. There is a small extra-anterior communicating artery.

From the left internal carotid, more toward the median line than the anomalous posterior communicating, there is a small artery (the anterior perforating artery), figured but not named in Heitzmann's *Anatomy*, p. 441, as coming off of the posterior communicating artery, which runs across the left optic nerve and dips down into the brain substance alongside of the left *corpus albicantia*, where there is an *extra, raised, pin-head* spot of cerebral substance. There is here a distinct aneurism filled with clotted blood, the greatest distention being in the centre, where it is dilated ten to fifteen times the size of the artery at its exit from the middle cerebral and its entrance into the brain substance. It tapers off, therefore, at both ends.

Dr. Deaver had received the impression that it was the speaker's view that the existence of marked arterial degeneration indicated the existence of syphilis. If this had, indeed, been the speaker's view, he must enter his dissent; for though syphilis is known to be a factor of great importance, he was certain it had been overestimated by many. Even in young men other causes must be sought and excluded.

Dr. Meigs also dissented from the opinion of the speaker, and pointed out that he had studied the arteries in many cases, and had found most advanced sclerosis in the absence of syphilitic history, as well as healthy arteries in cases of marked syphilis. The so-called "syphilitic endarteritis" he had come to discredit entirely.

Dr. Cattell, in answer to the gentlemen above named, stated that he had been led to the diagnosis of syphilis more by the history of the case than by the lesions. *June* 22, 1893.

IV. THE ORGANS OF RESPIRATION.

1. *Specimen of verminous pneumonia in a Rocky Mountain sheep.*

Exhibited by Dr. Frederick A. Packard.

The portion of lung from which the section presented was cut was sent by Dr. C. B. Penrose to Dr. Caspar Morris, for whom I have made the examination. I received a piece of tissue of a slate-color, rather heavy, and of the size of a hazelnut. The surface was somewhat irregular, and upon pressure the mass showed but little elasticity. Upon section the cut surface was found to be coarsely porous, with thickened septa. Imbedded in celloidin the mass was found rather difficult to cut owing to the tendency of thin sections to crumble before the knife.

Under the microscope the first field contained some bodies that looked almost precisely like *Trichina spiralis*. Further investigation, however, showed that there were in other fields bodies that were of fairly regular oval shape, with smooth outline, and filled with granular material; and still other bodies, fragments of which occupied either the minute bronchi or connective-tissue cyst-like cavities. The lung tissue itself showed thickening of the septa and a catarrhal exudate in the alveoli. The parasite was evidently a specimen of the *Strongylus ovis-pulmonalis*.

The following description of the parasite and its life-history is abstracted from the report of Cooper Curtice upon *The Animal Parasites of Sheep* (U. S. Department of Agriculture, Bureau of Animal Industry), the acompanying figure being borrowed from the same work.

The male measures 16 mm. in length, the female 25 mm.; in width the male measures 0.5 mm., the female 0.17 mm. The female is oviparous with two uteri and ovaries. The eggs segment after being laid.

The young escape from the lungs of infected animals and are scattered over pasture lands, yards, etc., in this way being apt to infect other sheep by ingestion with the food or drink. In some way they reach the bronchi and become encysted. In the cyst they attain the adult size and become sexually mature. The containing cyst ruptures, the now

mature parasites enter the smaller tubes and mate, the eggs are laid, the young are hatched out and enter the neighboring air-vesicles. It is unknown how long a period is required for the completion of the life-cycle and whether the intervention of an intermediate host or growth in other media is required.

The section herewith presented shows three phases in the life of the parasite—the egg, the young worm, and the mature individual. The specimen would, therefore, throw no light upon the question of the

Sections of lung affected by strongylus ovis-pulmonalis. (CURTICE.)

3, adult worms in bronchi; 4, young worms in alveoli; 5, segmenting eggs in alveoli; 6, developing embryo in alveoli; 7, *a*, embryo of strongylus filaria; *b*, embryo of strongylus ovis-pulmonalis.

necessity of an intermediate host; but it would seem that the only phase of life that is unaccounted for here is the growth from young to mature worms. Whether the young worm in this animal could have become sexually mature in its present host is, as has been stated, still unknown.

The occurrence of the parasite in the lung of a wild animal—one therefore that would be supposed to be but little apt to become infected—is very interesting.

The accompanying illustration exactly reproduces the appearances seen under the microscope save for the absence of a complete adult individual in my sections. *December* 10, 1891.

2. *Epithelioma of the face; extension; death from aspiration pneumonia.*

Exhibited by Dr. C. W. Burr.

These specimens were taken from the body of a man who died at the Home for Incurables to-day. The history of the case is very meagre. The disease first manifested itself as a warty outgrowth which was repeatedly cut off in shaving, and gave no trouble for several months. Then there occurred a somewhat rapid spread to the mouth, constantly increasing in extent, until feeding by a tube became necessary and had been practised some time before death. Death occurred after a brief febrile attack, and at autopsy the following conditions were determined: intensely marked bronchitis of the left lung with engorged, crimson and, to cursory examination in places, almost hemorrhagic mucous membrane; solidification of the left base, evidently from aspiration pneumonia; and right-sided bronchitis without pneumonic complication. The kidneys are those of a chronic Bright's disease.

March 10, 1892.

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3. *Acute tuberculous broncho-pneumonia.*

Exhibited by Dr. S. Solis-Cohen.

The lungs which I show were removed from the body of a colored man who died recently at the Philadelphia Hospital. Clinically there had been fever somewhat resembling in its course that of typhoid fever. Dulness, bronchial breathing and subcrepitant râles were found at both bases, later pleural effusion in the left chest, and finally dulness and bronchial breathing at the right apex. No definite history of the beginning of the illness could be obtained, but it seemed to be acute. Death occurred after a period of ten days. The diagnosis during life was acute tuberculosis, and two days before death tubercle bacilli were found in the sputum.

At the autopsy there was found well-marked tuberculous broncho-pneumonia involving both lungs. The racemose appearance of the tuberculous areas is well shown in the specimens. At the bases are areas much resembling, to the naked eye, ordinary croupous pneumonia. A small area of the same appearance is seen at the apex. The liver, spleen, and other organs seemed entirely free of any implication; but two of the mesenteric glands were enlarged and cheesy.

March 24, 1892.

4. *Encysted empyema.*

Exhibited by Dr. Henry W. Cattell.

Adherent to the left lung, just opposite the pericardium, was discovered the cyst which was shown to the Society, and which contained an enclosed cavity the size of a fist wearing a No. 6 glove. The contents were semifluid (cheesy), not filling the entire cavity, and measuring one and a half to two ounces. There was a distinct pyogenic membrane, and nothing resembling a dermoid was discovered.

October 13, 1893.

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5. *Gangrene of the lung from disease of the fibula.*

Exhibited by Dr. S. Solis-Cohen.

The specimens I show have unusual interest in calling to our minds the importance of carefully treating local conditions lest disastrous disturbances arise in other parts. In the case in question an inflammatory affection about the fibula—in itself not at all severe—gave rise to pneumonia, and subsequently gangrene of the lungs.

Only the portions of the *autopsy* record bearing on the pathological conditions named are appended.

Heart. Left cavities filled with soft, black clots. Left ventricle firmly contracted and hard. Right auricle contained yellow clots, some black clot and fluid blood. It was distinctly enlarged; the valves of the right side of the heart were normal. Coronary sinus easily admitted a small finger and did not show any valves. The muscle of the right heart was unusually firm, 12 mm. thick, pale in color, and the papillary muscles were distinctly mottled. Left auricle is normal. Mitral valve is somewhat thickened along the edge, but not retracted. The anterior surface of the anterior leaflet of the mitral valve showed several patches of atheromatous infiltration.

The left aortic valve showed a patch of calcareous infiltration at its base; other aortic leaflets were normal, and there were many small patches of atheromatous infiltration around the sinuses of Valsalva and around the coronary arteries.

The muscle of the left ventricle is about 23 mm. thick. Color of outer portion of muscle is brown; deeper layers are yellow; papillary

muscles are mottled and very pale; muscle is firm. The cavity of the left ventricle is not enlarged. The coronary arteries are extremely atheromatous.

Lungs. The left lung did not collapse readily; crepitant throughout; at apex there was a distinct fissure running at right angles to the fissure separating the lobes.

The right lung was the seat of general acute pleurisy; and the interlobular septa presented dense, thick, fibrinous exudate, which, however, was easily separated. The lower portion of the middle lobe showed a cone-shaped area of softening, the fluid of which was very dark-green in color and very offensive. This was evidently an infarct; it was about three inches in length and one and a half inches at the periphery. The lower lobe showed a similar condition in its middle portion, except that its length was not more than one inch, and gangrene had not progressed so far. The entire central half of the upper lobe was the seat of consolidation. There were two small infarcts in lower lobe, one of which was necrotic and the other not. There were several patches of pneumonia in the lower lobe, and the central lobe showed an extensive area around the gangrene. This lung was also extremely emphysematous, and the upper lobe contained frothy fluid.

An *embolus* was found at the apex of necrotic area of middle lobe in right lung—reddish, consistent, hard, 4 mm. in length, 3 mm. in width, 2 mm. in thickness, and attached to wall of pulmonary artery.

Stump. Right leg, a small ulcer over end of the fibula; section of the bone shows marrow red for one-half inch; normal beyond. Marrow extends to end of bone and is not covered by osseous tissue.

March 9, 1893.

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6. *Croupous pneumonia and parenchymatous nephritis.*

Exhibited by Dr. H. Toulmin.

The specimens presented were removed from the body of a man, aged 67 years; the body was well preserved; there was no history of alcoholism, syphilis, or other taint. The patient was admitted to the Presbyterian Hospital complaining of pain in the chest, cough, and frothy expectoration. There was considerable fever, and on physical examination consolidation of the left base and spots of blowing breath-

ing in the right lung were easily detected. The treatment consisted in the administration of carbonate of ammonium, digitalis, strychnine, oxygen, and finally blood serum, but the patient succumbed.

At *autopsy* the lungs were found to present these typical pneumonic appearances, and interesting conditions were seen in the kidneys; the left was greatly reduced in size, not as a result of congenital hypoplasia, but from atrophy, as the thin cortex and accumulation of fat within would prove. The other kidney was large, fatty, and presented evidences of parenchymatous nephritis. The latter is interesting from the fact that nephritis in association with pneumonia is probably much commoner than is believed. The urine was high colored, heavy, and loaded with albumin, but casts had not been carefully searched for.

March 9, 1893.

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7. *Tuberculosis of the lungs and serous membranes.*

Exhibited by Dr. M. V. Ball.

A 6031, male, aged 35 years, shoemaker by trade, spent the last twelve years in prison. November last he complained of anorexia, cough, loss of flesh, and has been sallow-complexioned and short-winded for the past year. Transferred to hospital. Fever—104° evening, 103° morning; slight expectoration; cough short and at night continuous. There was marked dulness over left lung, especially the middle portion, both anteriorly and posteriorly. No râles, and bronchophony was present. Family history phthisical. The sputum could not be obtained because of the very scanty amount. There were exhausting night-sweats.

Treatment. Cod-oil, quinine, expectorant mixtures, and liberal supply of food.

Two months later my notes state that fever and sweats continue. Appetite good; gaining slightly in weight; still no expectoration, but short hacking and annoying cough. Flatness now on left side. Fever—100° to 102°.

April 1st, he had lost twenty-five pounds. Severe dyspnœa for the past two weeks, but this was relieved by terpin hydrate, and is again on his feet and gaining.

May 30th, feels very comfortable and appears to be gaining in weight;

no fever; no cough; no expectoration; complains only of dyspnœa at night, and pain over right and left lung and over liver.

June 6th, was exposed to draught; complained of great dyspnœa; anorexia; emaciation became rapid. Fever slight, but profuse sweats, and on June 22d death occurred.

Post-mortem. Body extremely emaciated. Pleura adherent to walls of chest and lungs, and much thickened; at lower part fat-like clots of fibrin between it and the chest wall. Right lung enlarged, left lung one-half size; both filled with small, soft white nodules. These, when exposed on cover-glass and stained for tubercle bacilli, gave negative results. A small portion of lung teased out showed in unstained preparation nothing more than fibrous tissue filling the air cells.

The kidneys were congested. Spleen normal in size and appearance. Liver enlarged with capsule thickened.

Along the omentum and mesenteric covering of the intestines numerous small white points were found similar to those which covered the pleura and those found in the lung. There were no enlarged glands.

The sputum was examined some time before death. It was chiefly composed of saliva and mucus, and showed no tubercle bacilli.

The diagnosis *inter vitam* was fibroid phthisis.

The brain presented nothing unusual microscopically.

June 22, 1893.

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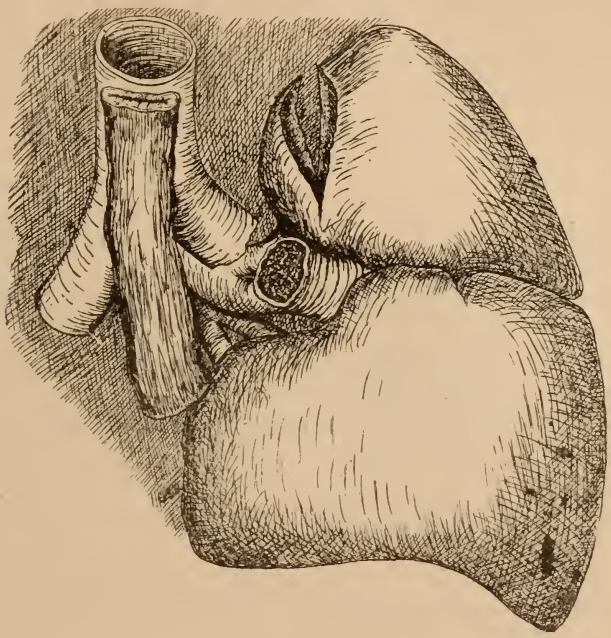
8. *Traumatic rupture of the lung and bronchus.*

Exhibited by Dr. G. G. Davis.

G. H., aged 22 years, colored, while driving a cart had his horse run away. He was violently thrown out, striking a tree. Exactly how he struck it was impossible to determine. On admission to the hospital his mouth and face were covered with blood, and he coughed and spat up bright red blood in large quantities. He was gasping, making great efforts to breathe, and continually threw himself from side to side. The rattle of the blood in the lungs and bronchi and trachea could be very distinctly heard. There was no evidence of any fracture of the bones of the chest or elsewhere. He died in about an hour.

On *post-mortem* examination the right chest cavity was found to contain a large quantity of fluid blood. The right bronchus was

torn completely across just as it entered the lung tissue. It was filled with clotted blood. A tear was also found in the upper lobe of the



right lung, extending upward from near the rupture of the bronchus. The rupture of the lung was two inches in depth, and began a couple of inches from the summit of the lung, and thence extended downward. I have no knowledge of any similar case. *June* 22, 1893.

V. THE GENITO-URINARY SYSTEM.

a. KIDNEYS, BLADDER, URINE, ETC.

1. *Kidneys from a case of gout.*

Exhibited by Dr. Allen J. Smith and Dr. F. S. Pearce.

Dr. Smith presented before the Society two kidneys removed from the body of a gouty patient in the Presbyterian Hospital. These organs were of normal size; the capsules were removed without special difficulty, leaving a surface slightly uneven. They had been surrounded by a considerable fatty deposit, and contained a notable amount of fatty tissue in the pelvis of each organ. On section of these kidneys there was noticed in the pyramids, mostly toward the apices, a very marked reddish deposit of crystalline nature, probably urates. The history of the case was as follows:

C. S., white, male, aged 46 years, married, a hotel clerk, and later a candy shopkeeper, was admitted to the Presbyterian Hospital May 22, 1891. Father still living, but has suffered a number of times from rheumatism; mother dead from apoplexy. Has several brothers living —none of the latter have had gout; an uncle and a grandmother have, however, had gout. The patient was always of a full habit, tended to sedentary customs, and usually preferred meat diet. His first attack of gout occurred nineteen years ago, the right middle finger becoming swollen and painful at that time. The present exacerbation occurred three weeks ago, when the right index finger became swollen, painful, and the seat of a deposit of urates in the first joint. Almost all of the joints of the fingers of the right hand were affected during the attack. The toes were not especially affected; there was a slight tenderness on the inside of the left ankle, and the knees were swollen and tender. Deposits of sodic urates could be readily picked out of the helix of the left ear. On admission the temperature was 100.6° F., the pulse 90, and respiration 26. The patient was constipated, but passed more than the normal quantity of urine, of highly acid reaction, specific gravity 1005, and precipitating a large deposit of uric acid and urates upon standing for a short time.

The patient was put upon anti-lithic diet and salicylic acid, the latter drug being presently changed for carbonate of lithium. On the third day after admission his bowels were freely opened, and sixty-three ounces of urine of very high specific gravity were passed. The following day there occurred a gouty exacerbation, the patient becoming delirious, the joints exceedingly tender, and the temperature increasing (104.8° F. on sixth day after admission). Potassium citrate and salicylate of sodium were given at this time, and the temperature began slowly and remittingly to decline until June 1, 1891, when it was again normal, pulse 92, and respiration 22 to 24. During the height of this outbreak the red blood-corpuscles were counted, numbering 4,800,000; there never was a lower proportion while the patient was under observation. The temperature remained normal from June 1, 1891, to June 9, 1891; the daily amount of urine during the same period ranging between 34 and 44 fluidounces, of a specific gravity of 1005 to 1010. The patient continued delirious; the joints remained tender; and presently the temperature took on a hectic type, ranging from 100° F. in the morning to 103.4° F. in the evening. The urine began to contain a trace of albumin, and increased considerably in the daily amount. The bowels tended to constipation, but were occasionally moved freely by calomel and magnesia.

The patient died in a comatose condition, with a temperature of 105.4° F., on July 1, 1891. *September* 10, 1891.

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2. *Renal anomaly.*

Exhibited by Dr. Allen J. Smith and Dr. R. M. Richards.

This specimen, as exhibited by Dr. Smith, consisted of the two kidneys of J. L., a white man, aged 61 years. The left kidney was larger than normal, weighing about eight ounces, lobulated, and presented on examination a thickened cortex, pale color, medullary portion not distinctly separate from the cortex, capsule non-adherent, subcapsular surface smooth—in short, a large white kidney. The right kidney was represented by a small mass of renal tissue about two inches in length, one-half inch in breadth, and perhaps one-quarter of an inch in thickness. It was covered with much fat, and was studded with small cysts. The pelvis of the kidney was a trifurcate one and quite enlarged, probably holding a fluidounce. The ureter was narrow but patulous. The kid-

ney felt very like a small, shallow sac with walls several lines in thickness. The exhibitor looked upon the organ as one whose condition was the result of hydronephrosis in early life.

The clinical history of the patient from whose body this specimen was removed bears little or no relation to the renal anomaly, although in an indirect way the latter may have had some influence upon the fatal termination of the illness for which he entered the Presbyterian Hospital, on January 21, 1891. His family and previous personal history were negative; and there was nothing to suggest in the slightest way any insufficiency of renal tissue. In the autumn of 1890 he had had influenza. On March 20, 1891, he experienced a severe chill, followed by vomiting and a cough without expectoration, and with a violent pain in the right chest. The respiration was rapid, the pulse quick, temperature moderately elevated, and the physical signs of a right-sided croupous pneumonia of the base were present. Death occurred after several days' illness.

It is probable that in this case the left kidney was in a condition of compensatory hypertrophy owing to the inefficiency of its fellow; added to this normal increase in size there is a pathological increase in the presence of cloudy swelling of the essential cells of the organ, due to irritation probably from the poisons developed by the acute disease, which were expelled with the urine. It is interesting to note in this connection, that in spite of this decidedly small area of renal vascularization there was no cardiac hypertrophy, the only change in the heart being a slight opacity of the mitral leaflets and a dilated and engorged right ventricle, the latter dependent upon the pulmonary condition. The renal circulation in this case cannot have been equal by at least one-third or more to the normal kidney circulation, and in accordance with the views of a large portion of the profession one should have expected a decided muscular hypertrophy of the left heart. It may be objected that such hypertrophy should not be found because of the early time of life that the right kidney assumed its small size. No matter the period of life, however, where there is obstruction to the arterial circulation, whether by occlusion of a notable portion or by pressure from without the vessel walls—in any manner, in short—the heart muscle invariably responds by a proportionate increase. It may be permitted, in support of this last proposition, to recall the case of cystic kidneys in a fœtus reported in the last volume of the *Transactions* of this Society (vol. xv., p. 184) by Dr. M. H. Fussell, in which the enlarged kidneys by pressure upon the abdominal aorta obstructed the onward passage of the blood, and in consequence of which obstruction the heart was

enormously enlarged on both sides, the right and left heart being in communication at this period of life. The absence of any cardiac enlargement in this case stands then in direct opposition to those authorities who claim that in the contracted kidneys of gout, and interstitial nephritis in general, as a necessary sequence there occurs left-sided hypertrophy; further, it is in so far an evidence that vascular changes, alterations in the vascular walls, are almost entirely the basis of such increase in cardiac muscular tissue.

As to the atrophic kidney, it may be said that usually where one kidney is atrophic it is the left; in the present instance the right kidney was the diminished one. There is no single cause to be assigned to the condition; it usually occurs, however, during intra-uterine life. In the present instance the dilated appearance of the pelvis and the sacculated appearance of the kidney strongly suggest a congenital hydronephrosis as causal. Examination of the ureter all the way down to the bladder did not, however, reveal any obstruction from growth, from injury, or calculous presence to account for the existence of the hydronephrosis, although it is entirely possible that in the 60 odd years of the patient's life such an obstructive change might easily have disappeared. In most instances, as in the present, under the microscope are to be discovered elements of hyperplasia, evidences of a low grade, chronic, formative inflammatory process begun in the embryonal organ, and arresting its development. *September* 24, 1891.

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3. *Cystic kidneys.*

Exhibited by Dr. H. F. Formad.

The first two specimens are kidneys from a man aged 77 years. The cysts in this case are due to obstructions in the uriniferous tubules. It is a very common thing to find the kidneys of old people in this condition.

The next specimen is from a case in which but one kidney was found. The cyst in this specimen was almost as large as an infant's head.

November 12, 1891.

4. *Congenital cystic kidneys.*

Exhibited by Dr. Alfred Stengel.

The kidneys which I exhibit were taken from the body of a demented old woman about 65 years of age, who had fallen down a flight of stairs and died of shock. A rather bad injury in the region of the left knee was stitched, and at the time of death, a day later, was in good condition.

The mental condition of the patient was such that no accurate information could have been obtained, nor had she been critically examined before the injury. After the fall, however, she was carefully examined, and a considerable swelling of the abdomen noted. The characters of this I cannot give you, but the resident physician found fluctuation in both flanks, and looked upon this as indicating ascites. The patient remained more or less delirious until death. No record of the urinary examination was reported.

The autopsy revealed nothing of interest excepting these kidneys. Both are of about equal size, nine inches long, six broad, and four in thickness. They are composed of densely aggregated, clear cysts, which look not unlike masses of clear, amber-colored jelly, enclosed in a thin transparent membrane. In size the cysts vary from an eighth of an inch to an inch and a half in diameter. On incision, in some of them a clear and soft, but not watery, material escapes; in others the contents are more watery. The pelvis of each kidney is very much distended, but no obstructien was found in either ureter. The latter were comparatively small and not of abnormal appearance. On incision into the kidney the greatly dilated pelvis is laid open, but nowhere can any true kidney substance be detected. Here and there it seems as if the septa between adjoining cysts might be composed of kidney structure, but this is not certain. Microscopic examination has not as yet been made.

These are excellent examples of the pathological condition first described by Virchow, and variously designated "hydrops renum," "polycystic kidneys," or "congenital cystic kidneys." As is well known, such pathological conditions were first described as of congenital origin, and are generally regarded as such now; but it must, I think, be evident that the condition of things which we found in this woman at autopsy could not have been present since birth. The usual history of these cases is that the child is killed in the efforts at birth, or later succumbs

from obstruction to the respiration by pressure. One or the other of these results would seem, inevitably, to have occurred, had these kidneys been of such size at birth. However, it is not impossible that a less marked degree of the same condition of things may have existed, and that subsequent growth of the individual cysts caused the enormous size we now see. Even had these early dangers been avoided, it is difficult to understand how the woman could have lived to old age with kidneys so evidently damaged. It is possible, of course, that glomeruli will be found in sufficient numbers in the septa between the cysts, but even then one could not well imagine kidneys of this appearance as functionally active. However, the weight of authority, the frequency of this condition at birth, and its infrequency later in life, force us to the conclusion that the condition is indeed a congenital one.

Nothing very definite can be said of the cause of these cysts. It has been suggested by Virchow, that any one or all of four causes may be at work, viz.: occlusion of the urinary tubules, renal apoplexy with cyst formation, overgrowth of cellular elements with subsequent absorption, or absorption of cellular exudate. The condition is not rarely associated with various forms of monstrosities, and a family tendency has been noted by a few observers. Thus Meckel twice found very large cystic kidneys in monsters. Also Virchow, Brückner, and Schupmann saw cystic kidneys associated with congenital hydrocephalus, hydrencephalocele, uterus bicornus, and excessive development of fingers and toes. As to the family tendency, Virchow records an instance of a woman who gave birth to four children thus affected, and Schupmann one in which it was thrice repeated. *November* 12, 1891.

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5. *Chronic nephritis; dilated heart.*

Exhibited by Dr. Thompson S. Westcott.

The patient from whom these specimens were removed came under my observation for the first time at the autopsy. He was 52 years of age, of good habits. A diagnosis of disease of the kidneys had been made, though it was stated that no albumin was detected in the urine. Death occurred by a slow cardiac failure.

At *autopsy* the heart was found greatly dilated and the cavities filled with chicken-fat and soft, red clots. There were some atheromatous patches in the aorta, but the valves were unaffected. The substance

was evidently fatty. The arteries throughout the body showed evident signs of atheroma.

The *kidneys* were decreased in size and granular on the surface; the cortex was diminished in thickness, the substance fatty, and there was a considerable deposit of fat in the pelvis. The capsule stripped easily.

The *liver* on section was nutmeg, considerably enlarged and fatty. The pancreas seemed also of abnormal softness. The omentum was loaded with fat.

I have submitted the specimens principally on account of the very markedly fatty condition of the heart muscle. I regret that I cannot give any more definite data concerning the clinical history of the case.

Dr. Packard: It is of great interest to note cases of this kind, in which albumin has not been detected, and in which autopsy reveals advanced disease of the kidneys. This is the fifth instance in which I have seen this occurrence within the year. The reason lies largely in the tests we used to demonstrate albumin. In several instances, in which subsequent history proved the presence of Bright's disease, I have found albumin by testing with trichloracetic acid when it was absent to the ordinary tests, and I feel confident that this is frequently the case.

The President: The presence of extreme cardiac dilatation in the absence of aortic valvular disease in this case is interesting and would merit consideration.

Dr. Westcott: No etiological factors of importance could be discovered which could explain the group of pathological conditions, unless an attack of typhoid fever four years ago be considered as having bearing on this case. *December* 10, 1891.

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6. *Cancer of the bladder and cervix uteri.*

Exhibited by Dr. F. S. Pearce.

Elizabeth F., aged 50 years; white; single; of good habits, but always a hard worker; was admitted to the hospital December 24, 1891. Family history: father died of heart disease; mother was killed; two brothers living and well. Says no "tumors" in family. Previous history: always has been a very healthy person excepting for infrequent bilious attacks. Has had the ordinary diseases of childhood and rheumatism twenty-two years ago, and from that time on suffered from dysmenorrhœa and menorrhagia until the climacteric was established at he age of 45 years.

Present trouble began last January (1891), according to patient's story, but probably it existed much longer. She had extreme dysuria and diuresis; the urine became cloudy and deposited much mucous sediment on standing a short time. Pain in back and loins running down both thighs; worse at night. Kept at her work with these symptoms until July 4, 1891, when she consulted a physician for the first time. At this time also, pseudo-menstruation came on at irregular intervals. Concretions of phosphates collected in vagina.

On admission the vulva was found to be very œdematous; vesico-recto-vaginal fistulæ existing. There was a villous growth all through the vagina. Patient much emaciated and cachectic. November 30th, peritonitis set in, from which she died December 8, 1891.

Autopsy. Body of an emaciated woman. Anxious expression. No marks of injury. Abdomen considerably distended, and on opening the same, much foul gas escaped. Purulent peritonitis existed, pus being found all through the abdominal cavity. Intestines were glued together by bands of adhesion. Bladder was filled with a carcinomatous mass (fungiform). Uterus small. Cervix eroded by cancerous mass. Rectum the same. No perforations of intestines into abdominal cavity found. Kidneys and heart normal. Microscopic examination proved the growth to be epithelioma. *March* 10, 1892.

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7. *Pyo-lithonephrosis of both kidneys with hypertrophy of the bladder.*

Exhibited for Dr. O. H. Allis, by Dr. F. S. Pearce.

James A., aged 63 years. Family history negative. Suffered from cystitis a year before there was any suspicion of stone. Cut for stone in the bladder by lateral operation August 1, 1890. Symptoms of cystitis disappeared and were absent for about six months. Almost a year from time of lateral operation a suprapubic operation was performed, necessitated by continued irritation of retained and new-formed phosphatic deposit, which could not be gotten through the urethra on account of his immensely enlarged prostate; a number of very small calculi lying immediately behind that organ out of reach. After the operation, by placing him on hands and knees to get drainage, small gravelly portions of stone were taken away for two months. A blunt, hollow trocar fitting tightly into a canula, furnished the avenue for daily irrigation of the bladder, and as an outlet for the urine, the opening being closed by an antiseptic dressing after each washing. Patient died

about six months after the operation, his mind being clear up to the time of development of the fatal uræmia.

Specimens. Both kidneys, as you see, have lost most of their parenchyma, are soft and flabby, containing large pus-sacs extending from the pelvis to very near the cortex, which is much narrowed, the organs being in advanced sclerosis, upon which was engrafted the present condition. Small, gritty gravel of phosphatic nature is found throughout the pus-cysts in both these disorganized organs. The ureters are dilated in their entire length to the admission of an ordinary sized lead pencil; and, from the very large bladder openings of these ducts, together with this contracted bladder with much hypertrophied walls (½ to ¾ inch thick) and enlarged prostate, it would seem that the pyo-lithonephrosis was secondary to the bladder trouble by a process of damming back. The nucleus for the original calculi in the bladder may, however, have come from the kidney. The bladder walls are very hard, and the organ would not hold more than four fluidounces. At the upper part you see the continued opening to the exterior through the abdominal walls. The prostate is hypertrophied. No malignant growths found.

As a point of treatment, Dr. Allis thinks death in this case was accelerated by the suprapubic operation, as this caused the bladder to shrink, throwing all its pus contents and calculi into the ureters and kidneys. *March* 10, 1892.

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8. *Congenital cystic kidneys.*

Exhibited by Dr. C. W. Burr.

A. B., female, white; single, teacher, aged 57 years. Admitted to St. Joseph's Hospital, April 14, 1892.

Previous health always good. Had never complained of any symptoms referable to the kidneys. Four weeks before admission she complained of cough and diarrhœa. She was then treated for laryngitis. Two days before admission she began to be slightly wandering in speech.

On admission her condition was as follows: Delirium deepening into stupor; marked hyperæsthesia. Respiration varied from 22 to 36 per minute, finally stertorous. Pulse 112 to 127, and later so weak that it could not be counted. Temperature, 100° to 103.4°. Odor urinous. Pupils contracted. No convulsion, but slight twitching of arms and legs. No paralysis. The urine was drawn and contained about one-

sixth its bulk of albumin. No tube-casts. It was dark in color and alkaline. During six hours one and one-half drachms were secreted. There was very slight œdema of the ankles. Died fifteen hours after admission.

Post-mortem. Large woman, with much subdermal and omental fat. No ascites. The kidneys were enormously increased in size. Both kept their original form and were lobulated. The entire mass is made up of cysts varying in size from a marble to a lemon. On section, some of the cysts are filled with a clear fluid; in others it is darker, and in some almost chocolate colored. The pelves are buried in the cysts. The ureters are normal. The bloodvessels are normal.

The left ventricle is hypertrophied. The valves are normal. No other lesions, except slight apical adhesions, were present.

May 12, 1892.

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9. *Cyst of kidney.*

Exhibited by Dr. Henry W. Cattell.

The specimen was removed from a child of five years. The left kidney was seen to bulge the peritoneum forward, and on removal contained a cyst fully three inches in diameter. It has shrunk a great deal in alcohol since removal and the clearly fluctuating character is no longer present. I wish to open it now to demonstrate the nature of the contents. These are peculiarly consistent, red and pulp-like. There is a distinct capsule.

Dr. Burr: It seems to me, Mr. President, that such concentration of cyst fluids is hardly possible, and from the appearances presented to us now I should be more inclined to regard this as sarcoma.

Dr. Cattell: I believe this still to have been an ordinary cyst, from the plainly marked fluctuation obtained and from the great shrinking which has occurred. *October* 13, 1892.

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10. *The albuminuria and the Bright's disease of uric acid and of oxaluria.*

Read at the Conversational Meeting, October 27, 1892,
by Dr. J. M. Da Costa.

The more we study albuminuria and Bright's disease the more we are struck with variations from the ordinary type, and the more evident it

becomes that advancing knowledge will separate still other groups than those with which we are at present acquainted. By their study we may learn that they not only have distinctive origins, but lead to significant pathological changes, and manifest themselves, even early, by recognizable clinical features. One of these groups that has attracted my attention for some years, and that is very inadequately known, is the one originating in excessive uric acid formation or in oxaluria, and it is to this group that I here desire to call particular attention, and to examine, in the light of experience with a number of cases watched for long periods, the beginning of the malady, the pathological conditions produced, the special signs which distinguish it, and the treatment that is most helpful.

It may best illustrate my meaning if I first describe a few cases that taught me the value of this kind of research, and that familiarized me with the differences which these particular forms of albuminuria and Bright's disease possess. One of the most striking of these cases may be briefly told.

Case I. A well-known professional man consulted me about four years ago, suffering from indigestion, marked flatulence, and constipation. He was very nervous, much alarmed about himself, and greatly annoyed by an irritability of the bladder. Albumin was found in small quantities in the urine, as were tube-casts of hyaline character. The albumin was most marked in the afternoon urine. Repeated examinations showed much the same thing, except that on one occasion hyaline and a few epithelial casts were seen without albumin being detected. The acid urine, the specific gravity of which was never below 1022, and frequently upward, often deposited quantities of brickdust. Extremely heavy sediments were noticed after drinking a glass of beer in the evening. The patient was very nervous about himself and despondent, and all work became irksome. A European trip and a carefully regulated diet, largely lessening the nitrogenized substances, did him great good. For two years there has been no trace of albumin in the urine. Previous to its disappearance, it was only detected by the turbidity with acetic acid and boiling, and was not found by nitric acid. In an examination made fourteen months ago by a professional chemist who had previously repeatedly found albumin, the urine is spoken of as intensely acid, as containing excessive amounts of uric acid, but without a trace of albumin or of sugar.

The patient consulted me recently again, being much annoyed by irritability of the heart and pulsation of the abdominal aorta. I found no organic disease of either. The second sound of the heart was sharply

defined; the temporal arteries were full and prominent, suggesting atheroma; indeed, I had so noticed them before, but the pulse did not indicate this, being small and of doubtfully increased tension. His nervous system was again disordered; he had many of the nervous symptoms of lithæmia. He dreaded going into crowds and coming into contact with people, and was much disturbed about his own condition. The urine was intensely acid, depositing urates, but it did not contain a trace of albumin, not even when boiled with acetic acid. The irritability of the bladder had passed away. Strict diet, chiefly of vegetables and milk, is again doing good. He is keeping the urine free by the use of Poland water or of Londonderry Lithia, and is taking systematic exercise in the open air. The circulation is being controlled by aconite, and his improvement is very marked.

In the case to follow, the symptoms were so much like those of ordinary Bright's disease, from which the patient supposed himself to be suffering, and the albuminuria was at first so decided and so persistent that it was only by a close study of the symptoms for some time that their true meaning was discerned.

Case II. J. H. M., about 45 years of age, suffered at one time from irritability of the bladder, due to a stricture which Dr. Keen cured by dilating. When first seen by Dr. Keen, who subsequently referred the case to me, the urine was found to be normal. Mr. M. was a very nervous man, greatly troubled with flatulent dyspepsia; indeed, his chief annoyance was the constant belching and the intestinal rumbling. He was also a most pronounced neurasthenic, easily depressed, and dreading greatly enlargement of the prostate gland, which, however, was not found to exist. Early in December, 1890, albumin was detected in the urine, of which an accurate examination was made, at Dr. Keen's request, by Dr. Coplin, with the result of finding marked amounts of albumin with hyaline and epithelial casts in an acid urine of higher specific gravity than normal, and exhibiting abundant evidence of crystalline excretion. I placed him on a strictly regulated diet, largely of milk and such green vegetables as he could digest, gave him occasional laxatives, directed the use of Poland water, and, off and on, he also took Basham's mixture. He slowly improved, the amount of albumin lessening and the specific gravity decreasing. A useful prescription proved to be papoid and soda, under which the intestinal indigestion and flatulency were greatly improved, so much so that he himself became encouraged. Further improvement took place by a visit to Capon Springs in the summer, during which he passed large quantities of reddish sand, as he called it. He never had dropsy.

About a year after I first saw him there was no albumin to be found in the urine on several tests. In truth, he felt himself so much better that he stopped his visits during the winter, keeping up in the main, however, his diet. He was never entirely free from his dyspeptic symptoms.

In April, 1892, he saw me again, having been subjected to very confining work, and being much annoyed by the belching and the other gastro-intestinal symptoms. His tongue was coated; he was constipated, and was once more very nervous. But an examination of the urine showed that his kidneys were not much deranged. There was, it is true, some deposit of urates, yet only the faintest trace of albumin, none at all with nitric acid, the slightest turbidity on boiling with acetic acid, and a very faint cloudiness when tested with picric acid. I put him again on a stricter diet than he had been lately following, and more like that which had been originally directed; ordered him phosphate of sodium and light diuretic waters, and insisted on systematic exercise. He again decidedly improved, and when I saw him in October not the slightest trace of albumin could be found in the urine, either with nitric acid or with picric acid, or boiled with acetic acid. The urine was acid, its specific gravity 1020. He was still troubled with flatulency, but not nearly so much, and he made the remark that when he was so disturbed his head felt free and he was able to work better.

We see, then, in these cases the symptoms of a malady in which digestive disorder occurs, and in which the disturbed nutrition manifests itself in the urine chiefly by the high specific gravity, the urates, and the presence of albumin and casts that are commonly thought to indicate Bright's disease. We frequently also have some body waste, indisposition to physical and mental exertion, fatigue easily induced, an intermittent pulse, cold extremities; and, from the side of the nervous system, the gloom, the despondency, the irritability of temper met with in lithæmics. Headache, too, and vertigo, may be encountered, though they are far from constant.

I shall now group a set of cases alongside of those just described, in which it is not so much uric acid and urates that we find, as oxalates. The symptoms are in the main the same. But we often have more intestinal indigestion, with very marked flatulency, and there is apt to be even a greater amount of nervous depression. We have, in truth, all the well-known symptoms of oxaluria that betray themselves not only in the digestion, but in the nervous system. Otherwise we find the same high specific gravity of the urine, with the deposit of oxalates in place of urates, although occasionally they are intermixed, and we

obtain the same results from sustained treatment, though more slowly. As I have given some illustrations of uric acid disorder leading to kidney complaint simulating Bright's disease, I shall add a few cases as illustrations of the oxaluria that leads to kindred affection. Case III. is a striking instance in a scholarly gentleman from Boston, and valuable because many of the analyses were made with great minuteness by the distinguished professor of medical chemistry at Harvard.

Case III. Mr. W., aged 35 years, has not been in good health since a college student in 1883. He has had frequent attacks of nausea and dizziness, and, at times, staggering gait; the latter symptom, however, did not appear markedly until June, 1888. He also complained of numbness in the fingers on the right side, and of headache. His eyes were closely examined, but no sufficient cause was found for the attacks, nor for the headaches. He had occasionally distinct seizures of vertigo, and pain at the base of the brain. The digestive symptoms were very marked, with loss of flesh, flatulency, and, at times, great mental depression. The urine analyzed in 1889 by Professor Wood, of Harvard, showed a great excess of uric acid and of urea, also oxalates and small quantities of albumin, but no sugar.

The urinary condition was much improved by living strictly on milk and Vals water. He took on an average three quarts of milk daily, and for a time the albumin disappeared. Indeed, a report in July, 1889, speaks of its absence as well as of the absence of casts, and only of its increased acidity and of the large proportion of urates. But the absence of albumin in the urine, even at this period, was not constant, for five reports of urinary analysis from the Harvard laboratory, made between March 12, 1889, and October 5, 1889, record albumin. They show acid reaction, increased urea and uric acid, albumin very slight, sugar and bile pigment absent, specific gravity 1027 to 1015 (1015 last examination), chlorides normal, earthy phosphates variable, alkaline phosphates normal or increased; considerable sediment exhibiting at different times excess of mucus, spermatozoa, calcic oxalate crystals, microscopic concretions, acid urate of sodium crystals, urethral epithelium, cells from prostatic region, an occasional blood globule, and renal casts with renal cells adherent, leukocytes, small round fatty cells; in night urine in some specimens casts from the straight tubules with renal cells adherent.

Further symptoms of the case noted were a feeling of constant chilliness, attacks of vomiting, lasting sometimes ten days, occasional but not frequent headache, and good knee-jerks. The first sound of the heart was weak, but there was no organic disease. The bowels as a rule were

constipated. Under strict diet, laxatives, and exercise in the open air, his whole condition improved very much.

Early in 1891 the symptoms became again more severe, the indigestion was once more marked, and the attacks of vertigo and the periodic attacks of vomiting returned, especially if his uncertain appetite had tempted him to eat freely for some days. There was extreme sensitiveness to cold water, and a feeling of hunger causing faintness. Insomnia and a sensation of cold, particularly at night, were very distressing. A urinary examination in February, 1891, records the urine as slightly acid, the specific gravity 1025, no sugar, small amounts of albumin, normal phosphates, normal urates, many large and small crystals of oxalate of calcium, and several epithelial and granular casts with some hyaline casts. He was directed to drink Poland and Saratoga Vichy freely, to take as a laxative Carlsbad salts, and to avoid sugars in his diet, and for a time also meats. He took chloride of ammonium, ten grains three times a day.

Gradually, but particularly after a strict diet, largely of vegetables and milk—to meat he himself had taken a distaste—living in the open air, and keeping up a course of laxatives, his digestive symptoms greatly improved, and all the oxalates disappeared from the urine, and with them the albumin, so that, while he remained a nervous man and often hypochondriacal, he regained his health, certainly as regards the kidney affection. For over a year there have been no tube-casts and no trace of albumin. The specific gravity in an examination of the urine, made in January, 1892, was recorded as 1015, the chlorides were normal, phosphates normal, urates normal; some epithelium from the kidneys and bladder, and amorphous urates were found, but no albumin and no sugar, no casts or blood corpuscles.

Having seen instances like this and bearing them in mind, I have been able of late years to diagnosticate them readily. I have twice within the last year, in cases that happened to be brought to me by the same physician, Dr. MacCuen Smith, recognized this particular form of albuminuria and told the patients that they would recover, as has fortunately proved to be a correct opinion. It may not be without interest to describe these cases, as they are very illustrative:

Case IV. M. A. H., aged 42 years, had influenza in 1889, and has not been in good health since. He has been losing flesh, and has been much troubled with giddy spells. When first examined in November, 1891, the tongue was found to be slightly coated, the breath heavy; the bowels were regular. He had a compressible pulse, a short feeble first sound, no headache, felt very listless and languid, was

easily fatigued, and subject to attacks of great mental depression. A dry cough existed. The urine was noted to be of specific gravity 1030, acid, to contain small quantities of albumin, but no bile or sugar. In the sediment granular casts were detected, vesical epithelium and granular débris and oxalates, but no oil, no blood, and no pus. He was placed on Rochelle salts as a morning laxative, directed to take Saratoga Vichy, to abstain from sweets, to use only moderate amounts of meat, and to live on green vegetables, fish, and oysters. He also took from time to time courses of two-drachm doses of Basham's mixture. Exercise was urged upon him. He began to feel so much better by living more in the open air that he gave up his business, and now he is a completely well man. The urine has become normal, has no trace of albumin or oxalates; he is in good spirits and considers himself in excellent health.

Case V. The case of B. T. M., seen in May, 1891, presented similar features. The symptoms of indigestion were very marked; the least exertion, mental or physical, produced great exhaustion, and he, too, had periods of great mental depression. The urine was of high specific gravity, and contained oxalates and albumin. Under treatment similar to that in the preceding case, both oxalates and the albumin disappeared. The urine, frequently examined for eleven months, showed neither oxalates nor albumin. In July of 1892, after considerable worry for ten days, both calcium oxalates and a trace of albumin reappeared; but a more active return to the treatment, which had been almost suspended, caused these to disappear, and Mr. M. expresses himself now as feeling better than he has done for twenty years.

From the general tenor of the cases the form of albuminuria with its particular features will have become apparent. Nevertheless, it will conduce to accuracy if the most striking of these be fully examined. First, of the dyspeptic symptoms. These are rarely absent, though sometimes they are very slight. The signs of intestinal indigestion with flatulency and stools of changed color are more common than the evidences of gastric disorder. Lack of appetite or capricious appetite is, however, not unusually complained of, as well as a feeling of fulness after taking food. The circulation is prone to be irregular, sometimes rapid, sometimes slow. The pulse is often weak and faltering, or intermittent. Rigidity of arteries, or even increased tension, which is said to belong to the uric acid diathesis as well as to the contracted kidney, I have very rarely noted. The hypertrophies and other cardiac lesions of Bright's disease are conspicuously absent. The same may be said of dropsy and of the eye lesions. Of neither of these have I observed a

single instance. A symptom worthy of note is the slight rise of temperature, particularly in the afternoon; and this teaches us to think of what the kidneys are excreting when we try to explain those slight but long-continued elevations of temperature, seemingly causeless, that we sometimes meet with. The prominence of the nervous symptoms is always very evident. Listlessness, fatigue on the least bodily or mental exertion, forgetfulness, headache, melancholy, sleeplessness, giddiness, may be severally, or may be all, encountered.

The characters of the urine are very significant. There is first the high specific gravity in a urine that is about the normal amount or a little scantier than normal. The specific gravity generally ranges between 1022 and 1028, but I have known it persistent at 1036. The urine, on standing, often deposits urates, sometimes even uric acid, very often mucus. In the deposit, in place of urates, or alternating with them, we may find with the microscope very many of the crystals of oxalate of calcium. The uric acid does not necessarily manifest itself by the deposits of urates. It is best to determine it chemically. This is done most readily by Haycraft's process. It is not always in excess in cases which have all the other characteristics of the group under discussion; but these are generally the oxalurics.

The total solids are increased. The salts in the urine other than the urates are in normal proportion, or vary but little; at times the chlorides are increased, and not diminished, as they are persistently in contracting kidney—a point to which Dr. Wolff, who made for me many of the analyses on which these remarks are founded, called my attention. The phosphates are normal or increased; urea is not deficient; it is of normal amount or increased. The relation of the urea to the uric acid is various. If we take the standard which Haig has established for uric acid, as 1 of the acid to 33 of urea, this proportion may be found to be unaltered, or the uric acid even relatively increased. In the cases with oxalates, the excretion of urea is usually much augmented.

The amount of albumin is generally small, but it varies much with the time of day. I found it mostly in the morning urine, or in the urine after breakfast; and with the ordinary tests it is at times absent, especially in the evening urine. But here, as in other cases of so-called intermittent albuminuria, there is this fallacy: The albumin is not really absent, but only greatly diminished; for it may often still be detected by the finer tests, such as by slightly acidulating the urine with acetic acid and then using picric acid, or by Tanret's test, or by the metaphosphoric acid test. It is not unusual for the albumin to be accidentally discovered, either by examination for life insurance (Case

VII.), or by chemists testing their own urine for comparison. I have known such instances; in one, the urine was tested as a control experiment in the use of metaphosphoric acid. There has now been no albumin present for over a year, though there are still oxalates.

Casts are scanty or altogether absent. In character they are hyaline, or epithelial, rarely markedly granular, never fatty. In a few instances (as in Cases III. and VII.) fatty cells have been reported as occasionally existing. Casts and albumin mostly go together. Yet I watched a case for ten years in which for a long period hyaline and epithelial casts were very often found in the urine of a gentleman suffering much from oxaluria, without any of the tests by heat or by nitric acid finding a trace of albumin. This case came under my observation before I had learned to employ any of the finer tests for albumin. Complete recovery took place, and ultimately death occurred from an affection of the liver.

When now we come to the question of diagnosis, we find the main difficulty is undoubtedly that there are cases of contracted kidney with most obscure beginnings, cases in which the amount of albumin is extremely small and at first intermittent, and so often absent that, as in one instance that was finally brought to the post-mortem table and found to have contracted kidneys, albumin was only noticed once in eight examinations.[1] But even in such cases we have the low specific gravity of the urine of the contracting kidney to guide us, and we do not observe the crippled organ passing off abundantly urea, urates, or oxalates; indeed, the urea is often much diminished. In advanced cases there is no difficulty in the differentiation. The retinal changes, the heart symptoms, with the low specific gravity of the urine in contracted kidney, are strikingly different. On this I cannot dwell too much. I have yet to see the first instance of the kind of albuminuria to which I am calling attention, in which the specific gravity was persistently low. To high arterial tension we must not attach too much importance as a means of distinction; for it also occurs from uric acid in the blood.

Another form of albuminuria that it may be difficult to distinguish from this albuminuria of mal-assimilation is the dietetic albuminuria. Now, in point of fact, a number of the reported instances of the latter are really not instances of dietetic albuminuria at all, but belong to the group under discussion, although they are not so recognized. In true dietetic albuminuria the albumin is only found in the urine when cer-

[1] Brennan: New York Medical Journal, July 4, 1891.

tain articles of food are taken, generally in immoderate amounts, such as cheese, eggs, or pastry. This albuminuria, too, has its special periods; it is particularly common after breakfast or a mid-day meal. It only occurs after the ingestion of food, and then very quickly, and, as the cases of Grainger Stewart[1] prove, it is worse after mental excitement and exercise, and is unattended by tube-casts. Very significant is it that the albumin is never found in the urine before breakfast, and that the albuminuria lacks the signs of disorder of the nervous system; significant, too, is the high specific gravity of the urine with the attending persistency of urates or oxalates.

The albuminuria of severe exercise, with which we have become familiar through the observations of Grainger Stewart[2] on football-players and on soldiers, and through the critical analysis of Senator,[3] is discriminated from the albuminuria of perverted nutrition we are endeavoring to elucidate by its occurring only after strenuous exertion, and by the absence of all other symptoms as regards the digestive state and the nervous system. Undoubtedly, violent exercise will also in the albuminuria under discussion increase the amount of albumin temporarily. But it does not do so in a distinctive or peculiarly striking manner; and, the strain over, the abnormal ingredient shows itself in its usual way.

One more point let me mention in the diagnosis of the albuminuria of uric acid and of oxaluria. We must be careful, when we obtain reactions showing but traces of albumin, to be sure that it is really albumin and not mucin we are encountering; for the amount of mucus in the heavily loaded urine is often very great. With reference to the kind of albumin that is discharged, it is, I believe, serum albumin. But whether it may not be mixed with globulin, or this preponderate, is a matter for future chemical research, as well as in how far peptones also occur and become a source of error.

The form of albuminuria under consideration may be met with at any age. It is rare among children or old persons. It is common in growing boys. It affects the sexes in an extraordinary disproportion. Bright's disease, as is well known, is much more usual in men than in women. But in the albuminuria and the Bright's disease of uric acid or of oxaluria the difference is still more striking. In the many instances I have met with there is but one woman, now a young married woman, who had had the disorder for some years, and was cured by

[1] Lectures on Albuminuria, 1888.
[2] Ibid.
[3] Die Albuminurie, 1890.

going to Carlsbad. Even her case was not entirely free from uncertainty, as there had been a severe attack of scarlet fever a few years before the albuminuria was discovered. Still, irrespective of this case, I cannot doubt that the disease may occur in women, for some of the reported cases of intermittent albuminuria in women, as, for instance, the cases spoken of by Dubreuilh,[1] admit best of this interpretation.

I have just mentioned the frequent occurrence of the disorder in boys. Indeed, since I have been studying the subject, I have become convinced that the albuminuria of adolescence, the form so common at or for some years after the age of puberty, is in the majority of instances the albuminuria of uric acid and of oxaluria. Long before I understood this disease I was struck with the fact that the albuminuria of adolescence showed marked debility, digestive disorder, albumin in varying amounts, mostly small, urine of high specific gravity abounding in urates or in oxalates, or in both. The intermittent character of the albuminuria and the sparse tube-casts are also very suggestive. Let me add that I believe most of the so-called intermittent albuminurias, or functional or cyclic albuminurias—for such are the many names given to the same disorder in which albumin unconnected with a real disease of the kidney appears at certain periods in the urine—whether they occur in adults or in young persons, to be also instances of this kind of albuminuria, and that it is the tissue-waste, with its uric acid or oxalates, which causes in its excretion temporary disturbance of the kidneys. Is not also the so-called physiological albuminuria of similar origin? And does not the irritation of the kidney from the excretion of the tissue-waste have as much to do even with the albuminuria after forced exercise, seen in the contests of pedestrians or in soldiers, as the altered blood-pressure and active congestion the exercise produces? It may be, then, that, studied in the light of attending tissue-waste and disordered nutrition, more or less permanent, all these forms of albuminuria will receive a new meaning.

It is not my purpose to examine here into these different types of functional albuminuria further than is necessary to connect them with the object of this paper. But there is one point that experience has taught me which it may not be without value to mention. The cases of intermittent albuminuria in adolescents have been supposed to be due to a weak circulation which attends the languor and listlessness in some of the boys it affects. I cannot think that any disordered flow of blood in the renal vessels from weakness is the true cause. Indeed, one of the

[1] Revue de Médecine, 1887, vii.

most typical illustrations of the malady I have known was in a college student with hypertrophy of the heart and slight mitral disease, with whose case I have been familiar since his childhood. The circulation has been always over-active.

CASE VI. B. W., aged 19 years, a student at Yale, has had for years hypertrophy of the heart with mitral regurgitation, which followed rheumatic fever in 1883. Examinations of the urine had shown nothing abnormal to exist until January, 1892, when he saw me on account of frequent nose-bleeding. His tongue was somewhat coated; he did not complain of headache; the heart's action was about the same as usual, though the pulse was not quite so strong; he expressed himself as not feeling vigorous. The heart's impulse was still forcible, and indicative of the hypertrophied condition. I found albumin in his urine, and a subsequent examination of the evening and morning urine, made by Dr. Wolff, gave these results:

	Morning.	Night.
Appearance	Clear	Clear
Color	Yellow	Yellow
Reaction	Acid	Acid
Specific gravity	1030	1032
Total solids	6 per cent.	6.3 per cent.
Urea	2.8 per cent.	2.6 per cent.
Chlorides	1.7 per cent.	1.6 per cent.
Albumin	Faint traces	Traces
Sugar	None	None
Sediment by microscopical examination	Calcium oxalate copious.	Calcium oxalate copious.

In a number of specimens examined no casts could be detected. The enormous amount of crystals of oxalate of calcium pointed to the incomplete suboxidation that was going on. The urea, it is seen, was below the proportion of total solids and below the proportion of the chlorides. The patient was placed upon muriatic acid, laxatives, and diuretic waters, at first Buffalo Lithia, and subsequently Poland water. He was restricted in sugars and meats, and was allowed to take moderate exercise. On the 15th of January no albumin could be detected in the morning urine. On the 19th of February the specific gravity of the evening urine was 1038, and there were heavy deposits of urates and small quantities of albumin. In the morning urine of February 20th the specific gravity was 1030, the urine was acid, and no traces of albumin could be found by boiling with acetic acid, or by the nitric acid contact test, or by picric acid dropped into urine acidulated with acetic acid and boiled. From this time on a number of examinations of the urine showed no albumin. The treatment was, in the main, kept up,

occasional short courses of Basham's mixture substituting the muriatic acid. The diet was adhered to, and he was allowed moderate amounts of milk. A gradual improvement took place; the specific gravity decreased; though in several specimens it was still 1026; and since March 23d, while oxalates and urates have been found at times, there has been no albumin, and he has felt well. The last examination of September 26th proved him to be in good health, and he shortly after resumed his work at college.

The prognosis is favorable, though the cases may be of long duration, lasting several years, with occasional reappearance of the albumin in the urine. When recovery takes place, which I think is almost invariable under proper treatment, and albumin and casts have long ceased, it is not unusual for small cells still to be found with the microscope in the urinary sediment, very likely incompletely formed cells from the tubules of the kidney. Yet I believe that occasionally, from very long continuance of the disorder, fibroid changes may take place and interstitial nephritis result. Notwithstanding some doubts that suggest themselves, owing to the history, I think this to be a case in point.

Case VII. Mr. C. L., aged 53 years, a manufacturer, became aware of having a renal disease on applying to a life insurance company in June, 1882. His application was refused on account of albuminuria. Except that he was tired from strenuous attention to business, and was a dyspeptic, he regarded himself as in perfect health. The discovery of albumin in his urine led him to put himself under treatment, and to retire from business. He was actuated partly in this by the fact that he had lost two brothers by Bright's disease. In March, 1883, as the records of a very accomplished physician in Richmond, who examined him, show, he was suffering a great deal with lassitude and general depression, and complained of pain in the back and head, indistinctness of vision, and was rheumatic. The urine at that time had a specific gravity of 1020, the amount voided in the twenty-four hours being forty ounces, and contained 6 per cent. of albumin by measure; no sugar, but granular casts, torulæ, and fat-cells. It was acid, and showed 320 grains of phosphates in the forty ounces; the urates were not determined. By June 11, 1883, the patient being under treatment, the albumin as well as torulæ had entirely disappeared; so had the casts, and the phosphates were normal; the specific gravity was 1014.

The urine, in March, 1886, examined by the same physician, is recorded as having one-seventh per cent. of albumin by measure or bulk, and again granular casts; and to be of specific gravity 1014, and acid. The phosphates were half a grain to the drachm.

Mr. L. subsequently went abroad and consulted a very eminent London physician, who looked upon his case as one of albuminuria largely due to nervous injury and indigestion, and to renal gravel. His analysis shows the urine to have been of specific gravity 1016, acid, normal in urohæmatin, in oxalic acid, in hippuric acid, in hydrochloric acid, in sulphuric acid, but with an excess of uric acid and of phosphoric acid; there were no bile acids. There was a little albumin. There was none in the urine at 8 A.M.; some was found at 11 A.M.; at 10 P.M. it was again absent. Under the microscope torulæ were detected, but no casts. The urine, examined several times after this record, showed no albumin or casts, but always torulæ. The patient's health had improved very much under diet and treatment for his indigestion. Mr. L. consulted me September 27th, of this year, on account of severe headache coming in spells in the morning, and great annoyance from flatulency. The bowels were slightly costive, the tongue lightly coated; he complained of pain over the left kidney. The pulse was 80, of rather high tension, but the second sound was not accentuated, nor was there cardiac hypertrophy. A urinary examination showed the urine to be of specific gravity 1013, to contain a trace of albumin, a deficiency of urea, no sugar and no casts, though a good many epithelial cells were found. He was placed on a diet chiefly of milk, green vegetables, and fruit. Phosphate of sodium was given to him in the morning, and, subsequently, ten drops of dilute muriatic acid three times a day. His digestion improved decidedly under this treatment, and the headaches lessened greatly, yet an examination of the urine on October 20th still showed albumin, quite well marked with the picric acid test, and the nitric acid ring test. A subsequent and careful analysis by Dr. Wolff gave in the urine of a total quantity of 64 ounces in a day, the following:

Total quantity as given, 64 ounces (normal 48 ounces); appearance, slightly cloudy; color, yellow; odor, normal; reaction, very acid; specific gravity, 1015; total solids, 3 per cent. = 960 grains in twenty-four hours (by Vogel, normal 930 grains); urea, 1.7 per cent. = 518 grains in twenty-four hours (by Vogel, normal 542 grains); uric acid by Haycraft, 0.141 per cent., in twenty-four hours 43.2 grains (normal, 11.7 grains); chlorides (as NaCl) = 0.68 per cent., in twenty-four hours 206.4 grains (normal, 255 grains); albumin (computed dry) in twenty-four hours, 7.6 grains; sugar, none; sediment consisted of epithelial cells (bladder), also a few leukocytes and red corpuscles.

This proves that there is little, if any, diminution of urea, but a marked diminution of the chlorides. The uric acid, as determined by Haycraft's method, is about three times the normal amount, and the

ratio of the urea is as 12 to 1, which is a great increase, if we accept the estimate of Haig as to 33 to 1 being the norm. The sediment consists of ephithelial cells from the bladder, also of a few leukocytes and red corpuscles, but no casts are detected.

What is the pathological state that gives rise to the albuminuria of uric acid and of oxaluria? I think essentially a congestion of the kidney, with, should the hyperæmia persist, slight local inflammatory changes in the vascular cortex from the irritating effects of excreting increased amounts of ill-formed or broken-down tissue in the shape of urea, urates, or these imperfectly oxidized, as oxalates. In other words, the primary fault is not in the kidneys, though the kidneys suffer because they have to throw off the irritating waste products. Anything that lowers vitality and disturbs the nervous system may increase the faulty assimilation, and, with it, the irritation and congestion which produce the albuminuria. The question whether this kind of albuminuria ever passes into true organic affection of the kidneys has been already considered. I believe this to be possible, but of extremely rare occurrence, and, when it happens, to be brought about by the constant irritation leading to slow fibroid changes and gradually developing interstitial nephritis. It is held by some that all, certainly most, cases of Bright's disease originate in uric acid excess acting locally. This view seems to me too sweeping, for, even for the production of the gouty contracted kidney in which we have uric acid playing so important a part, we have other elements to take into account—the general changes that occur in the body, the state of the abdominal ganglia, and the condition of the arteries, which, in the various relations of Bright's disease, have been so admirably described by Gull and Sutton and by Arthur Meigs.

In the treatment of the albuminuria of uric acid and of oxaluria we must bear in mind that we are dealing with a special disease, one in which mal-assimilation is the main element, and the kidney affection only a conspicuous expression. The treatment must not be, therefore, purely that of Bright's disease, as we commonly meet with it, but largely that of the underlying state, a state chiefly one we are familiar with as lithæmia, or as oxaluria. The lines of treatment are, it is true, in the main parallel. Thus it is just as essential in this form of albuminuria as in the ordinary forms of Bright's disease to lessen the work of the kidneys by calling the various emunctories into play. Nay, it is more essential, for the kidneys have not only usual work, but largely more than their share of work thrown on them; and it is this extra work that leads to their damage. Their labor can be much lessened,

and the blood condition and the character of the excretions altered, by close attention to diet—which is, in the main, also that of Bright's disease, though it is stricter as regards the ordinary carbohydrates, and, on the other hand, does not insist so much on milk. Vegetables, especially the green vegetables, and fruits are freely allowed; tea, coffee, and cocoa are permitted, if sweetened but slightly; so are limited amounts of oatmeal, of buckwheat and corn cakes, of rice, of bread and butter; and so are oysters and fish. The white meat of poultry and of game may be moderately taken, but the meats containing much nitrogen, such as mutton and beef, ought, as a rule, to be avoided. I find, however, where considerable exercise is indulged in, particularly by growing boys, that meats may be eaten in restricted amounts without disadvantage. Milk I do not find especially useful, and direct only a pint or two daily. It is, indeed, quite unnecessary to confine the patients to a milk diet, as is so often done, and done with advantage, in the usual nephritic kinds of Bright's disease. In fact, I have known an instance where a permanent cure was effected on a vegetable diet, in which an exclusive milk diet had left the urine still albuminous. Eggs need not be forbidden, if they be not taken constantly, and if the yellow rather than the white be eaten. As regards sugars, I am always strict, for I have too often noticed how they aggravate the symptoms in lithæmics. Beets, for the same reason, are forbidden. Among vegetables, too, I never allow the rhubarb plant, for I have observed, after its use, oxalates, and with them albumin reappear. Salt may be always freely taken.

It is, as in all forms of albuminuria, important to keep the kidneys flushed. The free use of pure water, plain or aërated as fancy dictates, and of the mild diuretic waters, such as Poland water, Saratoga Vichy water, or Vichy water, is always of service, Drinking hot water at bedtime acts in some persons very beneficially as a diuretic, and gives a copious flow of urine in the morning—doubly serviceable then, for Roberts and other observers have shown that uric acid is largely excreted during the morning hours. Drinking fluids before or soon after breakfast is valuable for the same reason. Alcoholic drinks ought, as a rule, to be avoided, or, if used, to be restricted to light wines, not acid. Beer is forbidden.

The use of baths, not too cold, followed by systematic skin friction, is clearly indicated; and of the value of exercise in the open air I have seen many striking examples. As long as it is not excessive and too long continued, it does good service. It helps to oxidize the food-stuffs as well as the waste products, and produces free action of the skin.

Far from increasing the albumin, as violent exertion will, it diminishes it. I learned this lesson from experience. I examined the urine of a young man that was albuminous in the morning, and, after his taking a horseback ride in the forenoon, the albumin had disappeared. The good results seen from life in the open air led to the inquiry whether mountain air was beneficial, even in high altitudes. I have found it so, unlike what we so often notice in the ordinary varieties of Bright's disease. The urine improves, and the albumin may, for the time, cease to be voided. Sea air does the general health good, but sea-bathing I have known to increase the albuminuria, as has also been observed for other forms of the disease. Cold douches to the spine I have not systematically tried. Grainger Stewart speaks of their causing the albumin to disappear in dietetic albuminuria. It is, I think, important that the patient should not make much exertion in the early part of the day. In one of Clement Dukes' very instructive papers on "The Albuminuria of Adolescents"[1]—a form which I have already cited as belonging, I think, in the main, to the group I am here describing—he mentions as a significant sign that boys affected with it fainted when obliged to stand during morning prayers in chapel. Young people who have the complaint ought not to be pushed in their studies. It is in them an expression of a disturbed development of the growing organism; an expression of weakness of nerve force and of poor assimilation during growth.

Among medicines, laxatives are very important—phosphate of sodium, cream of tartar, Rochelle salt. An occasional blue pill, or a grain or two of calomel preceding the saline, is also to be recommended. So is a course of muriate of ammonia, or of iron, from time to time—iron, in the shape preferably of the tartrate of iron and potassium, or of Basham's mixture. In the cases with oxalates, nitro-muriatic acid remains a standard remedy, and Haig,[2] in his recent very interesting work, strongly urges acid as clearing the blood of uric acid. We must not in any case overlook the heart. The heavy work thrown on the kidneys, the general ill-nutrition which comes from the poorly oxidized elements circulating in the blood, and the lowered nerve force, disturb the heart, and the irregularity it exhibits may have to be met by digitalis or strychnine. The inhalation of oxygen has suggested itself, but I have not as yet used it sufficiently to be sure of its distinct therapeutic value.

[1] London Lancet, December, 1891.
[2] Uric Acid as a Factor in the Causation of Disease, 1892.

The treatment has been described in this paper at some length, because we have it in our power to make this treatment effective, and, by applying it to a form of albuminuria that can be clearly recognized, to rescue cases which otherwise might really swell the list of Bright's disease, with the dreaded name of which they are commonly labelled.

Dr. James Tyson: I am sure that it is quite impossible to add anything to what Dr. Da Costa has said upon this very interesting subject. There are however, one or two points which may perhaps with advantage be dilated upon. The first and most striking symptom of this affection which impresses me is the high specific gravity—the invariably high specific gravity in the class of cases spoken of. It seems to me that the presence of high specific gravity in any urine is almost conclusive evidence that it is not from a case of Bright's disease, except in very acute cases or in those very advanced cases where, although there is diminution of the solids, the water is in such slight proportion that the specific gravity is high. It seems, therefore, taking cases presenting these lithæmic nervous symptoms, the urinary symptoms, and the presence of granular and even epithelial casts with small albuminuria, the presence of a high specific gravity comes in as a diagnostic feature of the greatest value in eliminating them from cases of true Bright's disease.

There is one symptom which was not alluded to to-night, although Dr. Da Costa has referred to it on other occasions, that is the intense headache which is constantly associated with these cases, coming sometimes in storms, as it were, with copious discharge of uric acid and relief of the symptoms.

Coming back to this matter of high specific gravity, it is singular how very frequently high specific gravity is associated with what may be called harmless albuminuria, and indeed I have heard one of our English friends, in connection with this subject, say that any urine with a very high specific gravity will be found to contain a little albumin. That is an exaggeration, but probably a close approximation to the truth.

To pass on to the interesting collateral subject of functional albuminuria, or the albuminuria of adolescence, in one particular my experience has not been quite like that of Dr. Da Costa—that is, in the presence of the albumin in the morning urine. In my experience it is generally absent in the morning urine of boys and in other cases of functional albuminuria. A number of years ago, speaking in this room on this subject, Dr. Pavy, of London, recalled his experience of a young boy seeking a situation in the English civil service, and thrown out on

account of the presence of albumin, and, having consulted a medical man, he was advised to take for examination the urine passed on rising, which was found free from albumin. The same ruse might be practised by a life-insurance candidate. In some cases the urine passed on rising has been free from albumin while that passed later has contained albumin. It is a singular fact that while food has something to do with albuminuria, it has not always to do with it. I have had under observation cases of functional albuminuria, where the urine on rising was free from albumin, but before breakfast, the patient being up and about, albumin would be found. This I have known to occur over and over again. The relation of food and exercise to these cases is an interesting one. Dr. Da Costa has mentioned the fact that sometimes exercise removes the albuminuria. So it has been in my experience. On the other hand, food, while I have known it to produce albuminuria, sometimes is without effect. A good deal depends upon the kind of food In one case which I reported some years ago, the taking of common beef essence was invariably followed by albuminuria.

I had not myself thought or carried the thought far enough, that these cases of albuminuria of adolescence were the same as the albuminuria of lithæmia. It has seemed to me that in these cases the specific gravity is seldom so high and the amount of deposit not so great. They may, however, belong to the same category of cases.

To come to the treatment, my experience has been precisely that of Dr. Da Costa, and my plan is to recommend the same sort of treatment, not placing the patient upon a milk diet, but allowing three or four glasses of milk. I have allowed green, and in fact all kinds of vegetables. I have not gone quite so far in regard to the carbohydrates. I confess that, as from a theoretical standpoint there does not seem to be the same objection to the carbohydrates as there is to the nitrogenous articles, I have in the great desire which these patients evince to get new things, allowed them plenty of bread and butter, nor have I restricted the sugars. I know that it is the custom of others to cut off these articles, but I have not found it necessary, nor can I trace any imperfect results to a failure to eliminate these articles, although my experience has not been as large as that of Dr. Da Costa.

Dr. J. H. Musser: I rise only to emphasize one important lesson that we should take home, and that is that in examination of the urine it is not sufficient to simply make the ordinary tests for albumin, but the investigation must be pursued still further. I think it important to emphasize this and call particular attention to it. As a result of the routine way in which this examination is usually made, the patient is

often condemned when it is quite unjustified by the condition of the urine.

One word in regard to treatment. I am not in accord with Dr. Tyson in regard to the carbohydrates. I wish particularly to exclude them. In this I try to follow out the teachings of Ebstein, who not only in these forms of albuminuria, but in all varieties of Bright's disease, lays great stress upon the exclusion of the carbohydrates. Bread, potatoes, and foods of that class, I am sure are harmful.

Dr. Lawrence J. Wolff: I wish only to call attention to the possibility of differentiating between the urine from the contracted kidney and from parenchymatous nephritis. With the ordinary ingestion of chlorides we find that in contracted kidney the chlorides are diminished out of proportion to the urea, while in parenchymatous nephritis there is a considerable increase of the proportion of chlorides to urea above that which is normal. When I find a constant diminution in the proportional amount of chlorides, I suspect contracted kidney.

Dr. S. Solis-Cohen: I might, in connection with this subject, refer to a case, that of a woman who has been under my observation for six or eight years, whose urine has persistently shown a high specific gravity and is rather low in quantity, notwithstanding the administration of diuretics and water, and is most of the time loaded with urates. At times there are oxalates as well. This woman has the symptoms of lithæmia. She also has intestinal indigestion, and paroxysms of headache are a distressing feature of the case. This woman has also presented the afternoon rise in temperature, and on this account I sought the advice of Dr. Da Costa. Having seen more of this case and of other cases, I have no hesitation in attributing the rise of temperature to nervous irritation due to the presence of uric acid. In this case albuminuria has recently appeared with a few granular casts and a few hyaline casts. Whether or not this case will turn out to be one of true gouty kidney, or whether, as I would rather hope, it is one of the class of cases that Dr. Da Costa has described, of course time must show.

I have seen another case in a woman. This patient exhibits not exactly the symptom to which Dr. Da Costa alluded, of fear of crowded assemblies, but she has a fear of going into the street alone. She will not go into the street without her husband goes with her. For this reason it is hard to get her to take sufficient exercise. The urine shows high specific gravity, varying presence of albumin, varying presence of casts, and the temporary disappearance of albumin under diuretics and laxatives, especially phosphate of sodium and mercurials.

I have seen still another class of cases, in which the temporary presence of albumin and tube-casts has been associated with the presence of uric acid, and in which I have been unable to diagnosticate organic disease of the kidney. One of these patients is a girl, 20 years of age. The urine varies in specific gravity, sometimes high, sometimes low; never dropsy; never any symptom which can be put down to the presence of contracted kidney; but, in addition to the presence of uric acid, the presence of tube-casts, and the intermittent presence of albumin in varying quantity, there are certain vasomotor phenomena which have been of a great deal of interest. There are at times transient flushes upon the face, there has been urticaria, there has been sudden loss of sight with accompanying symptoms, as though due to some disturbance of the bloodvessels of the retina, but this has passed away so quickly that opthalmoscopic examination has not enlightened us as to the cause. It is not a case of hysteria. In this case there is a family history of the uric acid diathesis and a collateral family history of diabetes. I do not know what the connection may be. The connection of the uric acid diathesis with diabetes and the relation of uric acid to these cases of albuminuria, seems to illustrate the clinical value of cutting off the carbohydrates from the diet. In the second case I have been able to tell that the woman has been eating candy by finding albumin in the urine after it has been absent for some time.

I might add that in the case in which vasomotor disturbances are observed the red blood-cells are from time to time present in the urine.

Dr. A. A. Eshner: In closing the discussion, I hope that Dr. Da Costa will say something about the changes that one is likely to find in kidneys of this type, if there are any, and advert to the rationale of the occurrence of the albuminuria.

The President: One thing strikes me; certainly there must, in the cases Dr. Da Costa has described, be some irritation of the kidney, and a question of interest is whether or not the effect of such irritation is likely to be permanent. Another thought is, would there not be likely to be recurrences of the affection in these people who are subject to uric acid formations and to oxaluria? I remember very well one man whose urine I examined many times for my father ten or fifteen years ago, who for two or three years had constantly, on and off, oxalates in his urine, with albumin and casts and, I think, blood. I still see him walking the streets apparently well. It would be interesting to know whether or not the irritation which exists temporarily produces a permanent effect, and whether the attacks are not likely to recur, and the

final result be that the patient falls into one of the more ordinarily accepted forms of Bright's disease.

Dr. J. M. Da Costa, in closing the discussion, said: With reference to the way in which this is brought about, I did not say much, because it is difficult to come to a definite conclusion. For myself, I believe that the irritation produces congestion of the kidney, slight exudation, and then secondary changes. There are some who believe that the albumin comes out of the blood, and that the kidneys are secondarily affected. That is not my belief. I distinctly think that what our President has said represents the truth—that it is a local irritation resulting from this excessive waste.

What has been said about the recurrence is strictly true. I know of one patient where at once all the symptoms can be brought back by taking ordinary rhubarb plant. At once it produces oxaluria, albumin, urates, and tube-casts. If there are indiscretions in diet or the nervous system is lowered by any cause, the disorder may come back, but by knowing this and by studying the clinical record, you can prevent the patient from having these attacks, avoid real organic disease, and save their lives. Some of these cases have been under observation for ten years, and nothing has happened. A number have been studied for six years. I think that it is a fair inference that where albumin remains absent for that length of time, no permanent kidney disease has resulted. There is one case in which I am still in doubt, but with that single exception I have not seen a case where permanent kidney affection has followed, and it is fully ten years since my attention was first called to the subject.

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11. *Case of hypospadia; enormous dilatation of bladder, absence of left kidney.*

Exhibited by Dr. J. Madison Taylor.

B. S., aged 3 months, was presented at the end of my clinic hour on November 16, 1892, and a hasty examination made by myself, assistants, and my surgical colleague, Dr. G. G. Davis, a photograph taken by Dr. Hand, the house physician, and the mother directed to bring the child back on the following day for further study. On the 17th the mother returned, but merely to report that death had occurred suddenly from exhaustion early in the morning. Hence, incompleteness in the history and description is to be ascribed to the difficulties engendered

by that event, all our energies being exhausted in petitioning for an autopsy, which was grudgingly allowed and instantly performed.

Parents healthy, and two older children in sound condition. This boy was vigorous when born ; breast-fed partly, but digestion quickly failed, and a multiplicity of artificial foods were tried to meet the increasingly troublesome constipation, which seems to have been largely due to mechanical obstruction by the tumor. Abdomen always large, growing rapidly in size till the end, crowding upon the diaphragm to such a degree as to cause great dyspnœa at times, but no convulsions till two hours before death. No history of other disorders.

Examination. Small, emaciated boy baby of a pasty, clammy, yellowish-green skin, dry mucous membranes, cold extremities. The abdomen enormously distended, springing abruptly from the line of the ribs and curving boldly down to join line of pelvic brim. Surface exceedingly dense, percussion note dull in the main, fluctuation elicited but not marked. The lumbar spine is out-curved by pressure of tumor, causing a marked posterior curvature. Hypospadias, incurved glans, orifice of urethra on site of proximal end of frænum.

Autopsy. Organs above the diaphragm normal. Stomach very small. Tumor found filling entire abdomen, crowding viscera out of place, which last are much shrunken and pallid. Pancreas, spleen, and right kidney present no peculiar appearance except pallor and flaccidity. Left kidney not found at all. Left renal capsule seems equal in size to the right one. Three large post-peritoneal glands, in size from a marrowfat pea to a hazel-nut, occupied a position which might be that of the left kidney. Rectum attached to the left side. Adhesions low in the abdomen and all about the tumor. Tumor consisted of a large sacculation of the bladder. Bladder contained over a quart of urine; specific gravity, 1009; pale, clear, with a trace of albumin; microscopically negative. *December* 8, 1892.

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12. *Cylindroids in the urine, and their significance.*

Read by Dr. Alfred Stengel.

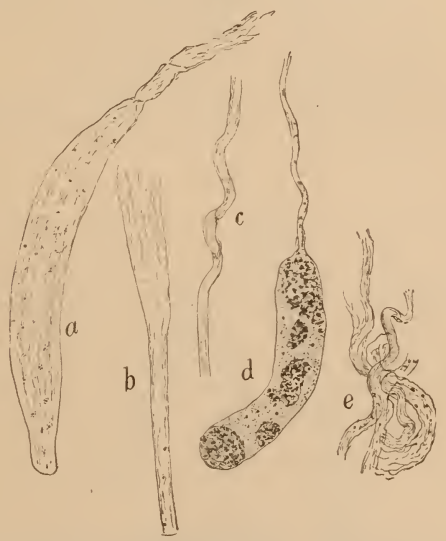
Cylindroids, so named from their resemblance to tube-casts, or "cylinders," are peculiar bodies first found in the urine of patients suffering from scarlet fever, by Thomas,[1] of Leipzig. Similar bodies had been previously noted by Meyer,[2] Lehman,[3] Frerichs,[4] Wyss,[5] Bock,[6] and Obermeyer,[7] in various conditions, but no accurate and systematic description had been published prior to the appearance of Thomas's paper. Subsequently, Pollak and Török[8] gave the subject careful investigation and experimented with animals to determine the nature and origin of these bodies. With the exception of these investigations, however, the question of the significance of cylindroids has of late received little attention.

The appearance of cylindroids is even more varied than that of true casts, but a regular succession of changes may be traced from one kind to others at first sight altogether different. It is possible, however, to distinguish certain types, which serve well for purposes of description, and we may consider them as of three forms:

[1] Thomas: Archiv für Heilkunde, 1870, ii. 130.
[2] Meyer: Virchow's Archiv, vi. p. 486.
[3] Lehman: Lehrbuch der Phys. Chemie, ii. 391.
[4] Frerichs: Die Brights'che Krankheit und ihre Behandlung, Braunschweig, 1851.
[5] Wyss: Archiv für Heilkunde, 1868, ix. 243.
[6] Wyss and Bock: "Febris Recurrens," Berlin, 1869, p. 150.
[7] Obermeyer: Virchow's Archiv, xlvii. 171.
[8] Pollak and Török: Arch. für exper. Path. und Pharmakol., xxv. 1888-89. Also, Bizzozero, v. Jaksch, and other systematic works.

1. The most frequent variety appears as thin, thread-like, or filamentous bodies, composed of transparent material, sometimes faintly striated, more frequently hyaloid, and always of considerable length. They may be traced for long distances across the slide and finally taper to a fine point or disappear from view by their extreme transparency. Sometimes they occur singly; more frequently they are in clumps in which the individual fibres are so massed together as to be inseparable from each other. Occurring singly, or even in small clumps, this form may be

FIG. 1.



Albuminous urine. *a*, *b*, and *c*. Ribbon-like forms. *d*. Cast-like form, with cells upon surface. *e*. Filamentous forms in a clump.

easily overlooked if too much light or too high a power is used in the examination. A lens of one-fifth inch focal length is to be preferred, and the light should not be central and intense.

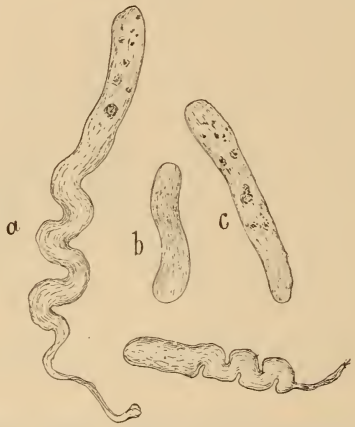
2. Next in point of similarity is a form that appears as ribbon-like structures, composed of material like the last, but more generally striated and evidently flat or "ribbon-like." They are frequently crooked or bent, and often tortuous. When bent at a sharp angle the edge of the cylindroid may be turned upward and clearly demonstrate the flatness of the body. The tortuosity is sometimes marked and appears simply as an exaggeration of what we are accustomed to see in some hyaline casts. The striations are fine longitudinal streaks, which are just distinct enough to give a general shading to the cylindroid without becom-

ing apparent as distinct lines. This peculiar, shaded, transparent structure is quite characteristic, when once one is accustomed to observe it.

Not rarely, this form presents the appearance of collapsed tubes of transparent material, not unlike, in this respect, the fibrinous casts of membranous bronchitis, though, of course, much more delicate and therefore completely collapsed. In size they vary from that of the narrowest to that of the broadest tube-casts, and they are sometimes much broader than any tube-casts I have ever seen. Like the filamentous form, they are long and occasionally, though more rarely, aggregated in masses. They may be forked or branched at the ends, and may even break up into a distinct leash, as seen in Fig. 1 (*b*).

3. A third, or cast-like form, is distinguished by its more marked resemblance to true casts, either hyaline or granular, and with this form

Fig. 2.



Non-albuminous urine. Cast-like forms, with deposit of urates.

I shall be mainly concerned in speaking of the importance of recognizing cylindroids. It is much shorter than the others, striations are more rarely present, and the appearance, as, indeed, the nature of the body, is rather that of a solid cylinder. The resemblance to hyaline casts is most striking, but these cylindroids may, in nearly all cases, be distinguished by the fine tapering end, or, if there is an abrupt ending, by the filamentous projection or tail, as is well shown in Fig. 1 (*d*). Unfortunately, however, it sometimes happens that the resemblance to a cast becomes complete by the absence of the tailed extremity, as may be seen in Fig. 2 (*b* and *c*) and Fig. 3 (*a*). In such case there remains

only the somewhat peculiar transparency or translucency to make the distinction, and this is a matter of doubtful value. I believe that there are certain forms that cannot be distinguished from true casts.

Fig. 3.

Non-albuminous urine. *a* and *b*. Cast-like forms. *c*. Filamentous.

Each of these forms is liable to alteration by the presence in the urine of various deposits, which may cling to the cylindroids, as they do also to casts. In cases in which there is considerable deposit of urates the latter may so cover the cylindroid as to produce the appearance of a granular cast, either pale or dark, according to the quantity of deposit. As a rule, however, the urate granulation is readily distinguished by the halo that surrounds each particle, and that is doubtless due to the refractive property of the granules. In other cases oxalate crystals are seen upon the cylindroid, and in still others epithelial cells or leukocytes, when the appearance is most strikingly like that of an ordinary cellular cast (Fig. 1, *d*).

Fig. 4.

Filamentous and ribbon-like cylindroids.

The recognition of cylindroids is rarely difficult, if ordinary care be taken. The filamentous form may be overlooked if too much light is

used, but, if seen, can scarcely be mistaken for anything else. The tape-like form might, under the same circumstances, be taken for a hyaline cast, from the fact that only a part of its length might be visible; but the peculiar striation, the bends and the tortuosity ordinarily make its recognition easy. The presence of clumps or masses either of this or of the last-named form would make the nature of the body still more manifest. In the third or cast-like form the difficulty, however, is considerable, and there are cases, as I have said, in which no distinction can be made. In these instances the abundant presence of other cylindroids of unquestionable type would incline one to regard the doubtful body as a cylindroid, though it must not be forgotten that cylindroids may be found in association with undoubted casts. If the body in question has a filamentous end or tail, I should not hesitate to class it as a cylindroid.

It might properly be asked what proof there is that cylindroids of the completely cast-like appearance ever occur, and on what ground they are distinguished from true casts. I can only answer that I have found in the non-albuminous urine of patients, not at all presenting the clinical manifestations of chronic nephritis, an abundance of cylindroids of the filamentous and tape-like form, and then an occasional one having the appearance represented in Fig. 2 (*b* and *c*) and Fig. 3 (*a*). Indeed, the two cases from the urine of which these figures were drawn strikingly bear out this view. In neither was there any albumin discoverable by cold nitric acid, heat and nitric acid, or the test with trichloracetic acid (which I believe is very delicate); neither presented clinical features to be ascribed to nephritis, and in each there were many undoubted cylindroids, with a few such as those here pictured. I am of the opinion, then, that there are cylindroids of this type whose nature and significance are similar to those of the other forms. This suggests the inquiry as to the possibility of such cast-like forms occurring unassociated with manifest cylindroids; but as we have no definite criterion by which they could be distinguished from hyaline casts proper, we should for the present be compelled in such cases to regard the structures seen as hyaline casts, whether the urine were albuminous or not.

The formation and origin of cylindroids were carefully considered by Thomas, and later by Pollak and Török. The latter instituted experiments on animals to determine the place and manner of their formation, and definitely settled the matter by finding them in the renal tubules, in sections of the kidneys of animals poisoned with turpentine and other irritants. The form alone would suggest this, without the definite proof. Their composition seems to be like that of true casts, from the fact that

they dissolve readily in acetic acid, as do casts. This test at the same time disposes of the view that they are composed of mucus, and the name "mucous cylindroids" is, therefore, improper. In some cases the cylindroids do not so readily dissolve in acetic acid, and Pollak and Török sought to show that such forms differ from the ordinary cylindroid in taking origin in the various tubular glands connected with the urinary mucous membrane, but this view yet lacks proof. Whether the cylindroids produced in the renal tubules are composed of substances derived directly from the blood, or of excretions of the epithelial cells, practically reopens the old discussion of the same question in regard to tube-casts, and the truth will probably be found in accepting both theories—the blood and the epithelial cells each take part.

Thomas, as well as later observers, conceived the possibility of true casts being formed by the folding together and fusion of cylindroids, and pictured forms like that shown in Fig. 2 (*a*) to prove this possibility. It seems probable enough that such an occurrence might take place occasionally or even frequently, but the matter at present lacks substantial proof. It is not unlikely, on the other hand, that a process of moderate activity might produce in the tubules hollow casts that, later, might collapse and form ribbon-like bodies; whereas, in more rapid and severe inflammatory conditions, an immediate output of excretory material might give rise to solid cylinders. All of this, however, is speculative.

The disease-conditions with which cylindroids have been found associated have been most varied, and it has been said that they occur in healthy urine. To the latter, Thomas objects that the patient or the physician himself might easily overlook some trifling affection, when in reality renal irritation could, by careful examination, be shown to exist. I have myself never seen cylindroids in urine that seemed perfectly normal in other respects. I have found them most commonly and most abundantly in urine in which there was excess of urates or oxalates; and since my attention has been directed particularly to their study, I have rarely failed to find at least a few cylindroids in such cases. As already mentioned, Thomas found them in the urine in scarlet fever, and after him other observers encountered them in various infectious diseases. Sometimes they occur in cases of undoubted nephritis before casts make their appearance; after the latter have disappeared in convalescence; or associated with casts throughout the case.

It will have been noted that in all of the diseases with which cylindroids have been found associated we have reason to think that there is renal irritation. In the infectious fevers, particularly scarlatina,

degeneration of the epithelium of the tubules is almost a constant feature; in nephritis it is most marked; and, according to the view of most clinicians, the presence of an excess of uric acid or urates occasions irritation of the lining cells of the uriniferous tubules, as of the blood-vessels. It is precisely in the class of cases that Da Costa[1] has recently described under the title of "The Albuminuria and Bright's Disease of Uric Acid and of Oxaluria," that cylindroids occur most abundantly, and in these the clinical evidence of renal irritation is certainly considerable. Of the pathologic conditions present in these cases Da Costa says: "I think [the condition is] essentially a congestion of the kidneys, with, should the hyperæmia persist, slight local inflammatory changes in the vascular cortex from the irritating effects of excreting increased amounts of ill-formed or broken-down tissue in the shape of urea, uric acid, or, these imperfectly oxidized, as oxalates." These conditions, according to my belief, are the cause of the occurrence of cylindroids; and the latter, I contend, do not occur in urine when such or similar renal conditions are absent. The presence or absence of albumin or its amount cannot, in most cases, be taken as a measure of the degree of renal irritation, and we find cylindroids when there is little or no albumin, as well as when there is a considerable quantity. In the infectious diseases the percentage of albumin is probably a better indication of the degree of irritation or disease, and Thomas found in scarlet fever, first, moderate albuminuria with cylindroids; later, more albumin and tube-casts; and, during convalescence from the nephritis, again diminishing amount of albumin and return of cylindroids, with absence of the casts, until finally all abnormal constituents disappeared.

In the cases of lithuria and oxaluria with which I have found cylindroids prominently associated, there have been some presenting a high degree of albuminuria and a larger number in which the presence of albumin could not be detected by any test. This point has already been referred to, and such cases as those from which Figs. 2 and 3 were taken have doubtless frequently been put down as instances of nephritis without albuminuria. I am sure I have done so myself in several instances.

It may be asked what advantage there is in separating cylindroids from casts proper, as it is admitted that both are formed within the renal tubules, are composed of similar material, and are indicative of renal irritation. Despite these facts there is much and good reason for maintaining a distinction. Of course, in a strict sense, these structures

[1] The American Journal of the Medical Sciences, January, 1893.

are casts or moulds of the tubules, but their form and appearance, excepting in the few cases referred to, easily distinguish them from the ordinary cast, and their significance is entirely different. Just as there is a great clinical advantage in separating the "albuminurias and Bright's disease of oxaluria and uric acid," which Da Costa speaks of, from ordinary chronic nephritis, in which the prognosis is so much more grave, there is an advantage in keeping the cylindroid, an indication of mild renal congestion and irritation, separated even in name from the nephritic cast, whose significance is more ominous.

If we could learn to feel that casts after all are not always an indication of serious renal mischief (as they undoubtedly occur in cases of transient renal congestion, icterus, and other conditions), it would be less necessary to retain the special name, "cylindroids." As far as the form of cylindroid that is almost or quite indistinguishable from ordinary casts is concerned, it is manifest from what has been said that there may be a regular succession of forms, from the marked and distinctive filamentous cylindroids up to such as are in reality true casts; but when the latter occur singly or in small number amidst numerous distinctive cylindroids, one cannot but view them with a considerable degree of composure. On the other hand, when hyaline casts appear in the urine in some number, without cylindroids, it would appear that the congestion or irritation producing them had been from the first more severe than such as ordinarily gives rise to cylindroids.

My conclusion is that cylindroids are formed in the renal tubules as a result of moderate renal congestion or irritation, that they may occur in albuminous or non-albuminous urine, and that they may be of such appearance as to be almost indistinguishable from ordinary casts, or may even be transformed into such. *February* 9, 1893.

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13. *Horseshoe kidney.*

Exhibited by Dr. C. A. Hamann.

This specimen was removed from a dissecting-room subject, an adult, male negro.

The fusion of the kidneys is at their lower border, by a bridge of renal tissue, two inches wide, which lay on the third and fourth lumbar vertebræ.

The left kidney is much larger than the right, and is flattened at its upper and anterior aspect to accommodate the spleen. The hilum of

each kidney is on the antero-lateral aspect of the organ. The pelvis of the left kidney, when distended, is almost as large as a hen's egg, and lies anteriorly, *i. e.*, it is in front of the vessels, the artery being the most posterior of the three structures.

The left ureter is about twice the size of a normal ureter; there was no obstruction to account for the large size of the left ureter and its pelvis.

The right ureter also lies anteriorly to the vessels; it begins as five or six calyces, which leave the kidney at different points, and fuse to form a rather large pelvis.

Each kidney receives three or four arteries from the abdominal aorta, which enter the organs at various positions.

The supra-renal bodies occupied their normal positions, showing that, as Hyrtl says, congenital displacements of the kidneys are not accompanied by displacements of the adrenals. *February* 9, 1893.

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14. *Pyonephrosis from stone.*

Exhibited by Dr. Joseph Price.

The kidney exhibited was removed by operation from a lady aged 60 years. There was a history of some sort of renal trouble thirty years ago, but she recovered and lived an active life until one year ago, when a growth was noticed in the abdomen, and eventually distended this cavity. It was difficult to distinguish from a large cystoma of the ovary, for there was no definite history of growth in an upward or downward direction. The growth, however, was immobile, while cysts of the ovary are usually mobile.

At operation this large cystic kidney was discovered. As is evident, it is an example of extreme pyonephrosis—the kidney being converted into a number of sacculated cavities, some of them very large.

Dr. Laplace remarked that the cysts in these cases are true retention cysts. *February* 23, 1893.

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15. *Stone in the kidney, with multiple purulent cysts.*

Exhibited by Dr. J. Hendrie Lloyd.

R. M., aged 50 years, white, a widow, was admitted to the Philadelphia Hospital on February 21, 1893, with a history of having had epi-

leptic convulsions. There was no history of syphilis or trauma. The father died of phthisis; the mother of paralysis.

These convulsions, it was ascertained, had first manifested themselves a year previous, and were ascribed by the patient to the menopause. They occurred at irregular intervals, sometimes thrice daily, again not oftener than once a week. In a severe fit, three weeks prior to admission, she had fallen and struck her left shoulder and elbow.

The patient was a large-framed woman, very dark complexioned, with timid, tearful countenance. She complained very seldom. During the first week in March she had a transient attack of gastric irritability, after the relief of which she seemed to be perfectly well. Toward the end of the second week in the month of March she complained of great fatigue after doing the light work in the ward which had been assigned to her. As she was an epileptic, and her intellect not of a high order, not much attention was bestowed upon her complaint; her temperature, morever, was normal. The only fact which caused comment was the peculiar rapidity of respiration (nearly 30), out of proportion to pulse and temperature. No satisfactory explanation of the phenomenon suggested itself, however.

On March 18th she became somewhat stupid, complained of pain in the abdomen, sat crouched up in her chair, and wept whenever accosted. Her answers were incoherent, the gait staggering and uncertain. She was put to bed and an examination made, but no positive results were obtained. On the following day her mental confusion was more pronounced—she had lost all idea of time and place.

The patient grew rapidly worse, her stupor deepened, and it became impossible to feed her. Death took place on March 21st, with the signs of pulmonary œdema. Seven hours before death the temperature was 99°, pulse 88, respiration 30. Four hours later, *i. e.*, within three hours of the end, the temperature was $103\frac{4}{5}$°, respiration 51, pulse 124.

An analysis of the urine had shown a large quantity of albumin, an acid reaction; color dark red; quantity of sediment large; pus-cells in great abundance; urates and uric acid crystals; no casts. Urine still acid. When this condition was found, the suspicion of uræmia was naturally entertained, and subsequent events proved that it was well founded.

Autopsy. The left kidney weighed twelve and a half ounces, being very much enlarged in all directions. It was soft and studded with multiple cysts, varying in size from that of a walnut to that of a peach. These cysts, on incision, discharged a thin, greenish, purulent liquid; and through an opening into one of the cysts a stone was felt,

and numerous small calculi were found in various situations and were easily felt from the surface of the organ. On cross-section of the kidney the substance of the organ appears almost completely destroyed, and the whole kidney is seen to be transformed into a multilocular cyst containing pus. The pelvis is occupied by a rather smooth, greenish stone, which is surrounded by fibrous and fatty tissue. The weight of the stone was 6.7 grammes. There were also a smaller calculus and considerable renal sand.

The opposite kidney is somewhat larger than normal, and presents on its posterior surface a small cyst filled with clear fluid. The cortex is slightly thicker than normal, and the vessels are prominent. The capsule is slightly adherent.

The lungs were œdematous. There was a small parovarian cyst.

April 13, 1893.

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16. *Bilateral double ureters ending in separate papillæ.*

Exhibited by Dr. Henry W. Cattell.

Double ureters are of rather frequent occurrence, though it is usual for the ureters to unite into a common trunk before emptying into the bladder. This union usually takes place in or above the middle third, though I have seen it occur in the coats of the bladder itself. The case about to be described is interesting, because upon each side of the body were found two distinct and well-formed ureters, not uniting in any portion of their course, but ending in the bladder in four separate papillæ.

The specimen was removed from a young Irish girl, aged 20 years. The notes of the autopsy are omitted, as they do not bear upon the congenital anomaly. The right kidney was normally formed, the two ureters of about equal size, starting from the union of separate calyces. At their origin the ureters were about an inch apart, but they rapidly approached each other and lay close together through the rest of their course, only being separated by loose areolar tissue. The wall of the bladder was thickened, and the papillæ were an eighth of an inch apart.

The left kidney was normal as to shape, one ureter coming off practically from its normal place, the other coming off by a separate pelvis below. They are nearer together than those on the right side, but the papillæ in the bladder are five-eighths of an inch apart.

I would like to call the attention of the Society to the old but useful and ever ready broom-whisp as a means of tracing canals. A nick is made above the pathological lesion or papillæ, and a piece of the broom is introduced and used as a probe.

I am indebted to Dr. James Wallace for his excellent photograph of the specimen. *May* 25, 1893.

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b. FEMALE ORGANS OF GENERATION.

17. *A series of gynecological specimens.*

Exhibited by Dr. JOSEPH PRICE.

Dr. Price placed before the Society a large group of gynecological specimens, and briefly referred to each in exhibiting them.

1. This was a large fibroid tumor, measuring perhaps six or eight inches in diameter, subperitoneal, firm, and of a pinkish color. It had been removed by the exhibitor in the previous July, in the West, from a young woman, aged 24 years. It had been of exceedingly rapid growth. The patient had had an attack of gonorrhœa shortly after marriage, and an abscess formed in the left side. This abscess grew rapidly, the temperature ran high, there was almost constant nausea, and her condition became alarming. Operation was decided upon, and this tumor, a large, firm fibroid, was removed. It is easily as large as an ordinary adult head. With this were also suppurating tubes, which were afterward removed as well. This associated condition is but another example of the frequent multiplicity of pathological conditions encountered in the pelvic cavity of a single case.

2. This specimen was another uterine fibroid tumor of about the same size as the above-described one. It was also removed from a young woman about 24 years of age, in whom it had been growing for two or more years. There were very tight and extensive adhesions, peritoneal and omental. The operation was marked by the free bleeding from these adhesions as they were separated. Dr. Price indicated the degree of hemorrhage when he stated he was compelled to use ligatures to check the flow of blood. He stated that he never uses ligatures in his intra-abdominal operations if he can help it, preferring the sponge packing. These adhesions the speaker urged before the Society as being the result of electrical applications employed with a view to cause the removal of the tumor.

3. The next tumor, also a uterine fibroid, was one of exceptionally rapid growth. The patient had returned from Europe, landing in this country only eight or ten days ago. In May last she started to Europe, exhausted after the removal of an intra-uterine polyp, following which an attack of septicæmia had occurred. At that time there had been no evidence of the tumors presented now before the Society. The specimen consisted of the uterus, enlarged, and containing in its cavity a tumor as large as a fœtal head. Besides, there was a second tumor in the wall of the fundus, another in the anterior, and a fourth in the left wall on the anterior margin.

4. The next specimen was a large ovarian cystoma. When it was of comparatively small size a practitioner who saw her advised the use of electricity for its removal. This was done without any good effect. On the contrary the tumor thereafter grew rapidly, almost leading to suffocation of the patient eventually because of its great size. Dr. Price, at the request of the patient, had several days ago removed the growth, which he found bound to all the surrounding tissues by firm adhesions, a condition which he would attribute to the use of electricity in such cases. Dr. Price expressed himself warmly as opposed to the employment of this agent in the treatment of ilio-pelvic new-formations as often leading to most pernicious results in exciting inflammatory reaction and adhesion formation.

5. This was a specimen of uterine cancer which presented no especial peculiarities, and which had been removed because of its hemorrhagic tendency.

6. This specimen was quite an unusual one, the only one of the kind Dr. Price has seen. It consists of a fusion of cystomata of both sides. There is a large cystoma of the right side, a smaller on the left. The tumors have grown, the one anteriorly, the other posteriorly, twisting the uterus about a half turn. The Fallopian tube on the right side is very long and large, measuring full fifteen inches in length.

7. This was an intra-uterine sarcoma. The cervix uteri in this instance was normal, which is not unusually the case in patients who are single and virgins. The speaker related, further, how some years since he had encountered an intra-uterine sarcoma in a virgin, and counselled hysterectomy. It was refused, and eventually a fistula was formed, leading from the lower bowel to the interior of the womb, through which fecal matter passed.

8. This was a small cystoma of the ovary, at the base of which there was a small papillomatous tumor. Two years ago the patient was aware of the existence of disease, and was examined. She shortly after became

pregnant, and operation was postponed. In the latter part of the pregnancy convulsions ensued, the pregnancy being brought to a termination artificially. Some time after this she returned to Dr. Price for operation. He found the cystic ovary with twisted pedicle, and a small ulcer close to and a little above the twist, and a minute papilloma at the base of the diseased organ. This last shows the characteristics of a soft papilloma under the microscope, as well as to the naked eye.

9. A collection of pus tubes, showing the large convolutions due to the enlargement of the tubes by the increasing pus within. Within the last four months Dr. Price has removed the tubes from as many as sixty patients. He pointed out the fact that where the fimbriated extremity of the tube is adherent to the ovary one may expect to find an ovarian abscess present.

10. This was a fibroid tumor of some size, pushed up and out of the pelvic cavity by an ovarian cyst. The tumor, though quite large, is of rather short duration, the growth having been rapid. Adherent to the walls of the ovarian cyst is the Fallopian tube, much elongated, its adherence by the fimbriated extremities marking the presence of an ovarian abscess. On the other side, in this case, there was a second smaller ovarian cyst.

11. This was an example of ectopic pregnancy successfully operated upon recently by Dr. Price. The tube was shown with a large clot of blood occupying it. The woman had complained, as is usual in these cases, of paroxysms of pain, followed by collapse. The abdomen was found full of blood, and the fœtal structures were not found. Dr. Price laid stress upon the fact that in tubal pregnancies the rupture of the tube usually takes place in the pavilion extremity of the tube, which is generally full of blood, and from which a large clot usually extends through the rupture. The speaker spoke of often finding the fœtus among the viscera.

In closing, Dr. Price took occasion to touch upon the question of pelvic inflammations. Granting the high authority of Simpson, who taught the existence of a pelvic cellulitis, Dr. Price must believe that in the primary stages of pelvic inflammation, at least, cellulitis does not exist. Only where there has been a breaking-down of the pustules, with a secondary production of cellular tissue, can he recognize its existence. Normally, between the layers of the broad ligament, there is very little cellular tissue to become inflamed.

Dr. L. S. McMurtry, of Louisville, Ky., present as a visitor, spoke by invitation at the conclusion of Dr. Price's demonstration. He said that he felt a hesitation in speaking before the Society as a stranger, but there

had been suggested so many things by Dr. Price's exhibition that he was prompted to make at least a few remarks. Referring to the fibroid tumors exhibited, he remarked that it was commonly stated in the text-books, and was moreover a very common popular and professional idea, that such growths occur more frequently among negroes than among white women. This belief he was inclined not to accept, judging from his own experience in a locality largely populated by the colored race. They seem to be quite as frequent in the white as in the black race. Nowadays there is held a question as to the propriety of operation in these cases; and it must be a common question what becomes of those cases in which the tumors are not removed. He felt satisfied that a much greater proportion die than we have any conception of. These tumors grow with much rapidity in many cases. They often produce death; from their size sometimes, becoming enormous, extending up to the ensiform cartilage, pressing upon the diaphragm, upon the stomach, the bowels, and interfering with their functions; pressing upon the kidneys and inducing an albuminuria, and thus eventually leading to death. "I mention this to you, Mr. President, as nothing at all new, but simply in order to state that fibroid tumors of the uterus must be looked upon as malignant, and that the sooner the idea that they are harmless, as is usually taught in a pathologico-histological sense, is abandoned, the better it will be for the human race.

"The operation for their removal is one of much difficulty, requiring the greatest skill; and as I witnessed the demonstrations of Dr. Price to-night, I thought of the change that has passed over the profession in these matters in recent years. Fifteen years ago such a series of specimens would never have been credited by the most advanced outside the reach of the operator's view.

"As to tubo-ovarian disease: one often meets with people in the profession who are constantly picturing as the most common of all affections of the pelvic region 'pelvic cellulitis.' For myself I have opened the abdominal cavity, and witnessed it opened by others, time and time again, and have never seen the cellular tissue *ab initio* the seat of suppurative processes. When it does occur it always follows some dirty thing—a dirty confinement, gonorrhœa, or the like. It is always to be treated by counter-irritation. This, with rest, generally makes the patient more comfortable. Eventually fistulæ are apt to form, and at best treatment, save it be surgical, is but temporizing.

"The case of ectopic gestation presented by Dr. Price illustrates the great advance which has taken place within the past few years in that department of surgery. I performed the first operation of this nature

done in Louisville, Ky., three years ago, and since then have performed it four more times. There is no doubt of the rapid education of the profession to the realization of these cases, even the recent graduate in medicine now being able and actually making correct diagnoses of ectopic pregnancies."

Dr. Price, in closing the discussion, said in relation to suppurative diseases of the tubes, he still meets patients ill and dying from these conditions. Often physicians smile at the statements of gynecologists about suppurating tubes; but he has often seen the greatest danger in such cases. For instance, to-day an invalid came before him for consultation in a most distressing condition from pus tubes—with sinuses in the groins, sinuses leading into the bladder, and as many as three to five about the sacrum. Death occurs from such conditions. One can scarcely conceive of a patient recovering without surgical interference from such a state. It is the exception to find a patient with any degree of general health, and it is usually said that they die of troubles other than these pelvic ones; but in reality the disease in the pelvis is responsible for these other troubles. *September* 24, 1891.

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18. *Pregnant uterus with fœtus intact.*

Exhibited by Dr. H. F. Formad.

The woman from whom these specimens were removed died in labor and while under ether. It will be seen that it was a face presentation.

The lungs were much distended, and on section into them vapor of ether escaped and was easily recognized in the room. The heart was dilated and full of dark, fluid blood. All of the organs were much congested. *November* 12, 1891.

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19. *Criminal abortion.*

Exhibited by Dr. H. F. Formad.

The womb from this case shows distinct evidence of injury inflicted by a lead-pencil which was said to have been used.

November 12, 1891.

20. *Polypoid intra-uterine fibroid.*

Exhibited by Dr. Joseph Leidy, Jr.

The uterus exhibited was removed by vaginal hysterectomy from a case of retroversion, with marked hypertrophy of the supra-vaginal portion of the cervix, decided prolapse, and troublesome vaginacele (cystocele or rectocele?). In the cervical canal may be seen a small polyp presenting nothing of particular importance, but illustrating the point that lesions which at one time seem of the gravest import, at another give rise to trivial or no symptoms. It is said that small polypi give rise to more serious hemorrhages than larger ones, but in the present case the woman of 38 years menstruated regularly and without excessive flow. *June* 9, 1892.

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21. *Bilateral pyosalpinx.*

Exhibited by Drs. J. B. Deaver and George Ross.

The specimens exhibited were removed from a woman, aged 28 years, who was treated in the German Hospital. The history obtained was as follows: Always healthy until six months prior to her admission. Her only child is 7 years of age; patient has not been pregnant since. General health good. Six months ago she believed herself to be pregnant. Menstruation ceased, morning sickness, abdominal pains. She suspected no trouble, and possibly would not have consulted a physician had she not fallen on the ice and sustained a Pott's fracture of the right leg, for which she was admitted. The following day she began to menstruate; but, with the history given, she was supposed to be about to abort. An examination showed no evidences of uterine pregnancy, but demonstrated a mass in the location of the right tube, very tender upon pressure. She denied any "bad disorder." The pain continued, the bleeding ceased, the temperature remained normal. Laparotomy was advised, to which she consented. Both tubes were found to be the seat of large collections of pus, the right being by far the largest. She is now convalescing rapidly. *January* 26, 1893.

22. *Miscellaneous gynecological specimens.*

Exhibited by Dr. Joseph Price.

1. The first was a specimen of ectopic pregnancy showing the ruptured sac and the fœtus. In presenting this specimen Dr. Price was led to remark on the frequency of ectopic pregnancy during the summer, he himself having seen six during the previous July and August, and then none at all during the entire winter.

2. A large pus tube of retort-like shape with an ovarian abscess. These pathological conditions in the speaker's experience are extremely common.

3. Carcinoma of the body of the uterus. This beautiful specimen showed a large fungating growth within the uterine cavity. The substance of the tumor was soft and brain-like, and it had invaded the wall of the organ to such extent that it in turn had become softened and had ruptured into the sigmoid flexure. Feces had escaped by the uterus and vagina. The cervix was entirely healthy and, as in so many of these cases which occur late in life, the canal was so narrowed that the cavity of the uterus could be reached in this way only with difficulty. Abdominal operation is preferable in these cases, but in any operation there is danger of breaking the very friable walls of the uterus.

4. Cystoma of the ovary. The woman from whom this cyst was removed had been delivered of a premature child at the sixth month. After the placenta had been removed by expression the cystoma was discovered, but gave no trouble until four days later, when there was acute pain, leaking of blood, and soon collapse. Operation was performed at once, and a cystoma with a twisted pedicle discovered. There were universal adhesions, but the tumor was removed and the woman recovered.

5. Carcinoma of the breast invading the ribs and finally the lungs. Nothing of interest was presented by this specimen excepting its penetration of neighboring structures.

6. Ovarian abscess with pus tubes. In these cases infection always comes through the tubes, and if there is a pelvic abscess it is invariably secondary to the tubal disease. *February* 23, 1893.

c. MALE ORGANS OF GENERATION.

23. *Hypertrophy of the prostate gland.*

Exhibited by Dr. HENRY F. FORMAD.

This enormously enlarged prostate presents on microscopic examinations typical cirrhotic appearances. We are, however, accustomed to call the disease inter-canalicular fibroma. Interest attaches to the present case from the fact that the cirrhotic condition of the liver, the granular kidneys, and the stiffened arteries, indicated a widespread tendency to fibrosis, and that the prostate in common with certain other organs is prone to be involved. *May* 12, 1892.

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24. *Epithelioma of the penis.*

Exhibited by Dr. JOHN B. DEAVER.

The man from whom this specimen was removed had been healthy and had led a moral life. He denied the possibility of venereal disease. The present trouble began a half-year ago, when he noticed a small pimple on the penis. This grew rapidly, and at the time of admission to the hospital it had attained the size of a goose-egg. The growth was typically cauliflowered, the penis indurated, and there was marked involvement of the inguinal glands. He had also tight phimosis.

The penis was removed close to the pubes, and as is seen in it, as laid open now, there is extensive epithelioma of the glans penis with involvement of the prepuce adjacent. *January* 26, 1893.

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d. MAMMARY GLANDS.

25. *Sarcoma of the breast in a boy.*

Exhibited by Dr. JOHN B. DEAVER.

The specimen exhibited really carries with it little of interest excepting in that it calls to mind the occurrence of sarcoma in the male breast. The patient was a boy of 17 or 19 years. He had not noticed any-

thing abnormal until a week before the excision. There was then a rapid growth. Microscopic examination shows the tumor to be a round-celled sarcoma. *December* 8, 1892.

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26. *Sero-cystic sarcoma of the breast.*

Exhibited by Dr. Frank Massey.

The tumor of the breast which I exhibit was removed by Dr. John Ashhurst, by whosekindness I am permitted to present it. The patient was a young woman of 24 years. Latterly she had gained much flesh; during the first year after her marriage as much as eighty pounds. Later the tumor was noted, and at first it would occasionally become inflamed and enlarged, subsiding to its previous size when the inflammation declined.

On removal the tumor was found to be encapsulated with a firm fibrous capsule. From this trabeculæ ran into its substance, which was of a fleshy character. Brodie divided adenomata into the dense, the cystic, and the cystoid, in which there are dilated ducts with cysts springing from them.

In this case there is no true glandular tissue, but only new-formed connective tissue. The structure of such growths very strongly suggests a parasitic cause, but such remains to be demonstrated.

February 9, 1893.

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27. *Multiple soft fibromas of the breast.*

Exhibited by Dr. Henry W. Cattell.

The tumors were multiple and removed from the breast of a woman. The one kindly sent me for diagnosis by C. W. Schmehl, of the third-year class of the University of Pennsylvania, was kidney-shaped, lobulated, encapsulated, and had a special blood-supply. Size about one and a half inches by one inch. The slide under the microscope shows it to be one of a soft fibroma. *May* 25, 1893.

28. *Scirrhus of the breast.*

Exhibited by Dr. G. G. Davis.

Mrs. E., aged 55 years, had been suffering for about six months before applying for treatment. She first noticed her left breast becoming larger and painful. The pain was referred to the arm. The axillary glands were markedly enlarged and the supra-clavicular ones slightly so. The breast was not markedly attached to the chest walls. Motion of the arm of the affected side was limited. The whole mass, including the glands up to beneath the clavicle, was removed from the breast incision, and a few slightly enlarged glands taken away through a separate incision above the clavicle. *June* 22, 1893.

VI. THE NERVOUS SYSTEM.

1. *Multiple carcinoma, involving the cerebellum.*

Exhibited by Drs. A. J. Smith and F. S. Pearce.

E. W., female, white, single, aged 59 years, was admitted to the Presbyterian Hospital, May 27, 1891. Father and mother died in patient's early life, from unknown causes; two brothers and a sister living and well. The patient has usually enjoyed good health, except that when young she had several mild attacks of rheumatism. Six weeks prior to her admission to the hospital, she was suddenly taken ill with severe and persistent headaches and malaise, and great weakness. Anorexia became a feature of the case, a feeling of weight and discomfort always following the ingestion of solid food. On June 2, 1891, she became stuporous, and slept most of the time. She took nourishment well, however; but on the following day she began vomiting from no apparent cause. On June 17, 1891, the following note was entered: The patient has been recently vomiting almost daily, lavage, the use of cocaine, and other remedies having no effect upon the hyperemesis. The vomiting to-day was so persistent that nothing could be retained upon the stomach, and the patient was fed by nutrient enemata. On June 29, 1891, one month after admission, nodules were discovered in each breast, adherent to the submammary structures, but painless. There had by this time developed a very marked cachexia. The patient complained of no pain, was extremely apathetic, and could be aroused from her stupor only with difficulty. She continued to lose flesh and strength rapidly, the cachexia becoming more and more marked constantly. The stupor continued, the patient being aroused with greater difficulty, until finally, a short while before death, absolute coma was present. An examination of the eyes by Dr. Perkins, on July 11, 1891, revealed the presence of "choked disk" in both. Besides the nodules in the mammæ there could be felt enlarged glands in the axillæ; but no other palpable centres of cancerous formation could be made out. The case was looked upon as one of multiple secondary cancer, arising from an unknown primary source. The woman died about the middle of July, and an autopsy was held by Dr. Stengel acting in the absence of Dr. Smith as pathologist, from which were obtained the following outlines:

The body was that of an exceedingly emaciated white woman, with post-mortem lividity over the back of the neck, trunk, and legs. In the right breast could be felt a hard growth, presumably cancerous, about the size of a fist, and in the left a smaller similar one. The skin over the right breast was pitted and puckered; and on removal of the glands they were seen to be the seat of dense growth, of a homogeneous-appearing structure, pinkish-white on the cut surface. These growths were non-encapsulated and showed a distinct zone of infiltration into the surrounding tissues. Opening the thoracic cavity, it was noted that the lungs were normal, save for slight œdema about the bases; the pleural membranes and sacs were normal. The heart presented slight hypertrophy of the left ventricle, but no valvular lesions. The tissue surrounding the roots of the great vessels was discovered thickened and matted together, dense, and resembling much the nodules in the mammary glands. The abdominal viscera were found in normal position. The stomach was ecchymotic, but otherwise normal, and the intestines were normal; the spleen small and dense; the gall-bladder moderately distended with fluid bile and the ducts patulous; the liver was the seat of a number of small nodular growths, the largest the size of a walnut. These nodules presented the same general appearance as the tumors elsewhere; they were mostly situated on the periphery of the organ, but there were a few in the substance as well. The kidneys showed no other abnormality than a slight degree of congestion; the adrenals were also normal. Mesenteric glands were not involved, but the post-peritoneal chain of lymphatics was enlarged, dense, and evidently the seat of tumor-infiltration. At the head of the pancreas there were several nodules of tumor-growth. The uterus and ovaries were healthy; to the left Fallopian tube there was attached a small cyst containing clear serum hanging free by a pedicle.

On opening the skull cavity the membranes of the brain were found free from inflammation. The cerebrum presented a slight amount of congestion; on the superior surface of the cerebellum to the left of the tænia there was found a dense nodule of new growth, apparently encapsulated and secondary. The brain matter about the nodule was slightly softened; no other part was involved. *September* 10, 1891.

2. *Sarcoma of the pia mater of the spinal cord in a cat.*

Exhibited by Dr. WILLIAM B. FENN.

The cat began to show signs of weakness in the posterior extremities, which increased until the animal became completely paraplegic. It is a curious fact to note that about a year before a dog on the same premises was affected similarly and a tumor was found in the cord. Unfortunately, that specimen was spoiled before a careful examination could be made. In the case of the cat an autopsy was obtained by the aid of a little chloroform. The muscles of the back were cut away and the vertebral column opened with bone-forceps. A tumor was found on the posterior aspect of the cord, apparently in the membranes, extending for about four inches from the middle of the cord backward. It was thicker in some places than in others, but formed a continuous saddle, covering the posterior surface of the cord. Under a low power the growth is seen to saddle the posterior portion of the cord, which is compressed. It begins between what look like layers of the pia mater just anterior to the middle of each lateral column, forming a wedge, apparently splitting the pia into two layers, the inner layer remaining close to the cord and the outer forming a limiting membrane to the growth. Just to one side of the posterior fissure these layers, which are very distinct at this point, are apparently united by fibrous tissue, dividing the growth into two distinct portions, like a pair of saddle-bags. The tumor consists of small round cells, closely packed together. The cord itself is much degenerated; no healthy nerve fibres can be seen. The striæ from the horns of gray matter to the periphery of the cord are plainly seen.

The President: We are beginning to realize more and more the importance of studies in comparative pathology, and it is well that this is so; for, doubtless, very many important discoveries may be made by the side-lights thus thrown on human disease.

Dr. Formad: This specimen calls to mind the frequency of sarcoma, especially the melanotic form, in horses. I have had the opportunity of examining a large number of such. Veterinarians do not regard these pigmented sarcomata of the horse as particularly dangerous.

October 22, 1891.

3. *Three fatal cases of cerebro-spinal meningitis, with autopsies.*

Exhibited by Dr. Herman B. Allyn.

I desire to report these three cases because they occurred immediately following a widespread epidemic of influenza, in which cerebro-spinal symptoms were among the most characteristic features of the disease. At such a time the possibility of mistaking the graver for the milder affection is much more likely to occur, especially when one is not on his guard. These cases may, therefore, serve a useful purpose in the way of warning.

Case I. The first patient was a man, aged 40 years, married, and engaged in running coal-cars at Greenwich Point. After working for twenty-nine consecutive days, he awoke, on the morning of February 1st, with a headache, which became worse, and after breakfast he was suddenly seized with severe pain in the right ankle. He went to Greenwich Point to get excused for the day, and on his way home—a long walk being necessary—he became so weak in his legs and feet that he fell down. He lay on the ground for three hours, unable to rise, and vomiting profusely, and was possibly unconscious part of the time. A passing driver roused him, and, between driving and walking, he managed at last to get home in an extremely prostrated condition. He complained of pains in his knees, ankle, back, and head, pain in the latter being most intense. He was delirious during Monday night, and between three and four o'clock the next morning became unconscious, with rigid limbs and clenched teeth. This comatose condition lasted several hours, and then gave place to delirium, from which the patient was never afterward wholly free. In addition to the local pains he had general hyperæsthesia. For a few nights the delirium was maniacal, the patient fighting with his attendants and jumping out of bed. Insomnia was persistent.

On Friday an herpetic eruption appeared on the lower hip, and on the right wrist there were some forty or fifty papules, which subsequently became pustules, and then dried up. The wrist was stiff, tender, and somewhat swollen. The fever was moderate, the pulse under 90, and the respiration about 20. There was marked tenderness along the spine, especially in the lumbar region and at the nape of the neck. There was never any opisthotonos, retraction of the head, or convulsions. The lungs and heart were free from disease. Constipation was obstinate; the urine contained the merest trace of albumin and no casts; the conjunctivæ were injected. Gradually a typical ataxic typhoid condition

developed, the temperature rose, the pulse and respiration became more frequent, delirium was persistent, and it was increasingly difficult to feed him. On February 18th he was completely comatose, with very high fever and moist skin; pulse, 160; respiration, 64. He died after an illness of eighteen days.

The autopsy was held the same evening by Dr. Jamison, assisted by Dr. W. S. Carter and myself. The heart, lungs, liver, spleen, and intestines presented nothing abnormal. There was only one kidney, and that was the seat of acute and chronic nephritis.

The brain itself appeared normal, except for some softening. The lymph spaces of the pia were distended, and the arachnoid and pia injected. At the base, particularly surrounding the medulla, the arachnoid and pia were inflamed, the pia being adherent to the medulla, pons, and cerebellum and to the upper portion of the cord. A small quantity of lymph was found, particularly around the circle of Willis and the anterior perforated space. The blood was fluid. No cultures were attempted.

Case II. The second patient was a healthy man, aged 28 years, who was called in to care for the first patient at night. After several days of this watching, on February 5th, he complained at dinner of having pain in his back. He then went out into the back yard and while there fainted. He regained consciousness, and went upstairs to bed, complaining of being cold and of intense headache, backache, and pains "all over." The pain in the head was agonizing. Explosive vomiting occurred twice. Delirium was sportive and jocular in type. He sang a comic song, got out of bed to obtain a glass of cold water, poured it over his head, and, after making an intelligent reply to a question, was supposed to be sleeping quietly. He was found dead at twenty minutes past four, his illness having lasted about three days.

In a very short time after death, the back, from the shoulders to the buttocks, was purple from effused blood. Rigor mortis set in very early.

The autopsy was held on the following day by Dr. T. J. Ellinger, assisted by myself. Only the brain and cord were examined.

The dura mater was adherent, though not tightly, to the skull. The pia was everywhere adherent to the brain. From about the anterior temporal convolutions posteriorly the lymph spaces in the pia were much distended with yellow lymph. The injection of the pia and arachnoid gave the brain a bright-red appearance, contrasting strongly with the empty blue veins. The brain itself was anæmic. The blood wherever found was black and fluid. The inflammatory adhesions were especially well marked at the base, over the medulla and pons.

The spinal canal was full of black, fluid blood; the upper portion of the cord was much softened. The spinal dura had to be stripped from the walls of the canal. There were some flakes of lymph upon the membranes. Gelatin test-tubes were inoculated with the lymph.

Case III. The third patient was a sister of the first patient. She was 48 years old. There had been, as far as could be ascertained, no communication between sister and brother. She went to bed on the night of March 6th apparently perfectly well, except that she complained slightly of pains in her joints. She had eaten very heartily of turkey. During the night she was seized with a chill, with vomiting and purging, but no one knew of her illness until about five o'clock the next morning. I saw her first about 4 P.M., March 7th. Vomiting and purging had ceased; she was extremely weak, with cold skin and feeble, frequent pulse. The conjunctivæ were injected and the pupils contracted. The patient had no pain, except in the forehead. She continued conscious and quiet until twelve o'clock midnight, when she became comatose; pulse, 160; respiration, 40. She died in the same condition at 2.45 A.M., her illness having lasted about twenty-four hours.

The autopsy was made by the Coroner's physician the same evening. All the organs were soft and friable, with acute parenchymatous degeneration. The stomach and intestines showed marked inflammation. The mucous membrane was inflamed and softened and appeared rough, as though stripped of its epithelium. The dura was moderately adherent to the skull. The pia and arachnoid were thickened, and the outer surface had a gelatinous appearance, that next to the brain being much injected, but not adherent to the brain over the cortex. The lymph spaces were full, the veins empty. There was an unusual amount of fluid in the brain, and the brain itself was much softened. At the base, the same lesions were found as in the other cases, but were not so marked.

It is remarkable that these three cases occurred practically in one family, and that all of the patients were adults. The first case progressed gradually to a fatal issue, lasting eighteen days. The second was fulminating, lasting only three days. The third presented all the symptoms of acute toxic gastro-enteritis, with collapse and death. It would not have been regarded as a case of meningitis but for the findings at the autopsy.

The diagnosis from influenza, with which cerebro-spinal meningitis was most likely to be confounded, can be made in most cases by noting the occurrence, at the onset of the disease, of chill, vomiting, and faintness, accompanied or followed immediately by headache and backache,

the headache being more frequently occipital than is the headache of influenza. Subsequently there are hyperæsthesia of the skin, stiffness and tenderness of the muscles of the neck, injected conjunctivæ, delirium, and frequently convulsions. Eruptions, most frequently herpetic or petechial, are absent as often as present. The occurrence of other cases of the same kind stamps the disease as epidemic cerebro-spinal meningitis. Coma sometimes alternates with delirium and usually succeeds it in lingering fatal cases.

The ages of the patients probably account for the absence of convulsions. *February* 11, and *March* 10, 1892.

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4. *Softening in the pons Varolii; thrombosis of the right internal carotid artery.*

Exhibited by Dr. John B. Shober.

The patient, a woman aged 75 years, had been in good health until her fifty-fifth year, when there had been a sudden hemiplegia affecting the left side. There was no loss of consciousness. At the end of three months she seemed restored to health, and remained so until she reached the age of 72 years. At this time she began to suffer with attacks of heart-failure, for which strychnine and digitalis were prescribed. Three months after the beginning of these seizures she had the second stroke. There was sudden vertigo, but no unconsciousness, and hemiplegia became immediately complete. The ocular conditions could not be studied on account of cataract. Sensation was also impaired on the left side, the patient being unable to distinguish between sharp and blunt points.

The clinical diagnosis at this time was embolism, but no organic cardiac lesion had ever been determined, and only a suspicion of aortic vegetations had once been entertained. Ten days after the stroke the facial palsy began to disappear, the tongue was protruded in the middle line and the knee could be elevated in bed. Obstinate insomnia, which could not be overcome by any remedy, suddenly supervened and finally led to a fatal issue by exhaustion.

At autopsy a few pleural adhesions were found binding the left lung to its pleura. There was no alteration of the aortic valve and only a little thickening of the mitral. The arteries were atheromatous. The liver was slightly fatty; the other organs normal.

Brain. The dura was adherent to the cranium. There was a clot in the right internal carotid just as it joins the circle of Willis. It was an eighth of an inch long, and was so firmly organized that it could hardly have dated from so recent a period as that of the attack which finally led to the patient's death. In the pons on the right side there was a spot of softening about the size of a large pea. As I take it, the latter lesion was the cause of the recent attack, and the organized thrombus the occasion of the attack which occurred seventeen years before.

Dr. F. A. Packard was of the opinion that the first attack was embolic in nature from the transitoriness of the symptoms, and called attention to the fact that where valvular disease is not detected, the arteries, as in the present case, may be found atheromatous and that portions of fibrin may be derived from this source and act as emboli. *May* 12, 1892.

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5. *Atrophy of the motor speech centre.*

Exhibited by Dr. C. W. Burr.

The patient was a female, aged 28 years. There was no history of any serious illness. At no time had there been any trouble in speech. For some months before death she was in the State Asylum for the Insane on account of melancholia. She died ten months after the onset of the melancholia, of phthisis.

The skull was symmetrical, its average thickness 4 mm. Diploic tissue diminished. The internal surface of the calvarium was smooth and free from depressions. The dura was non-adherent to the calvarium and its vessels and sinuses were empty. The pia-arachnoid was slightly adherent to the cortex of the right frontal lobe. On the left hemisphere in the region of the operculum was a depression about the size and shape of a walnut. It was bounded anteriorly and superiorly by the anterior and superior portions of the inferior frontal convolution; behind by the anterior portion of the temporo-sphenoidal lobe; its floor was made up by the island of Reil; below it was open. The ascending frontal and ascending parietal convolutions escaped, except the lowermost part of the former. The atrophied part, then, involves a large part of the inferior frontal convolution, including the operculum and the most anterior part of the temporo-sphenoidal lobe. It includes the motor speech centre. The remaining main fissures are normal. The entire brain is, however, small, weighing only forty-three ounces. The cerebellum is small in correspondence with the cerebrum. Its halves are symmetrical.

The interesting point in the specimen is the absence of aphasia. From the history we are justified in assuming that the lesion occurred either *in utero* or at least before speech began to develop. It is, of course, well known that local brain lesions, especially those involving speech, are, in the young, very prone to be recovered from; or, to put it more properly, one hemisphere can assume the functions of the other more readily in early than in later life. As to the cause of the atrophy in this case, I am in doubt. It may have been either a development defect, obstruction of the circulation, or meningeal hemorrhage. *November* 10, 1892.

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6. *Globular brain in a case of epileptic insanity.*

Exhibited by Dr. C. W. Burr.

Colored female, aged 61 years. Born at term. No injury during labor. Nothing wrong was noticed until the third year, when she began to have epileptic fits. She never developed mentally, but soon sunk into imbecility. At no time was there paralysis. She died while in the State Asylum at Norristown, of phthisis.

Autopsy. The scalp was thick and slightly adherent. Average thickness of the skull 7 mm. Diploë largely developed. Brain moderately soft. Meningeal grooves broad and deep. Pacchionian depressions shallow. Dura tense, lustrous; vessels full; inner surface smooth, slightly injected, slightly brownish in color. Pacchionian bodies few and small. In longitudinal sinus a small amount of fluid blood. The pia is clear, slightly œdematous; larger vessels full. Upon removal of the brain a large amount of cerebro-spinal fluid escapes. The *brain* is very œdematous; weight 38½ ounces. The cerebrum is markedly semi-globular. The apposition of two such brains, base to base, would make an almost perfect sphere. The transverse and antero-posterior diameters are almost equal. The principal fissures are all present, but the brain is simple in type. *November* 10, 1892.

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7. *Spinal cord from a case of pernicious anæmia.*

Exhibited by Dr. C. W. Burr.

I am indebted for the opportunity to show this specimen to Dr. Morris J. Lewis.

Unfortunately many of the notes of the case have been lost, and I

am only able to give the report of the patient's condition on admision to the Orthopædic Hospital and Infirmary for Nervous Diseases, in November of 1889. Middle-aged white woman; bedridden; skin sallow, with a pecular yellow tinge; no dark spots; pressure causes no change in color; her body has a peculiar musty odor; spleen not enlarged; area of renal dulness not increased; lungs normal. Apex beat is in the fifth interspace in nipple line; impulse is a trifle extended; a systolic bruit is heard over the body of the heart, and at the apex, at which point it is loudest; it is perfectly soft and low; venous hum in neck; slight general œdema; veins in arms are not very marked; no tenderness over sternum or clavicle; back very tender to touch; no periosteal nodes; no glandular enlargements in groins or axillæ; no increase of liver dulness.

Blood examination. Red corpuscles, 1,150,000; hæmoglobin, 25 per cent. Three weeks later examination showed: Red blood-corpuscles, 860,000; hæmoglobin, 12 per cent.

The urine contained pus and albumin, but no casts. She gradually grew weaker, vomited so frequently that rectal feeding had to be resorted to, developed an acute bedsore, and was at times delirious. Slight transient paresis of left face and arm. Death in coma. Diagnosis: Pernicious anæmia.

Autopsy. Subcutaneous fat rather abundant, orange-yellow. Abdominal and thoracic viscera normal. The marrow of the tibia was blood-red, and of the consistency of currant jelly. On microscopic examination quite a number of nucleated red blood-corpuscles were found. Absolutely no fat was present. Macroscopically the brain and spinal cord showed nothing abnormal.

Sections of the liver treated with hydrochloric acid and potassium ferrocyanide gave the following results, as described in Dr. Griffith's and my paper on "The Pathology of Pernicious Anæmia." "The cells of the outer and middle zones of the lobules were very full of iron pigment, the amount constantly diminishing in the inner zone as the central vein was approached, so that in the greater number of the cells of this zone there was no appreciable amount of iron present. The iron was in the form of minute irregularly shaped granules. No pigment could be found in the capillary bloodvessels or bile ducts. Besides the iron pigment a yellow, granular blood pigment, such as is frequently visible in sections of the liver, was discernible in parts of the sections, chiefly in the region nearest the central vein. The wall of the central vein was stained a blue-green color in the specimens treated with ferrocyanide, but no granular matter could be detected in it." After hardening in alcohol

and staining by Pal's method, and by carminate of soda, sections of the spinal cord present the appearances you now see. The anterior and lateral columns and the gray matter are normal. In the posterior columns there is degeneration throughout the cord. In the cervical region the process involves both the postero-internal and postero-external column, there being between the two a V-shaped area of normal white matter. The degeneration does not reach quite to the posterior commissure nor to the gray matter laterally. The degenerated area grows less in cross-section as we go down the cord. In the middle dorsal region only a small spot surrounding the middle of the posterior fissure is involved, and in the lower dorsal and lumbar region a microscopic area in the same situation. Nowhere is the degeneration complete. In every section there are many normal nerve fibres. Some stand out naked, without sheaths, and others are gone altogether, their place being occupied by a finely fibrous material. The walls of the bloodvessels are somewhat swollen, and look hyaline. There are in spots many corpora amylacea. *December* 22, 1892.

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8. *Multiple neuritis and malignant endocarditis.*

Exhibited by Dr. J. Hendrie Lloyd.

The specimens shown are from a patient who died in the nervous wards of the Philadelphia Hospital. The clinical history is briefly as follows:

Ed. O'T., male, aged 49 years, born in Ireland, married, an iron-worker by trade, was admitted to the nervous wards of the Philadelphia Hospital on April 5, 1893.

This history was obtained largely from his wife, the patient's own statements being confused and contradictory.

Family history. Negative.

Previous history. Always healthy previous to present illness; a hard drinker; no history of syphilis; has always worked very hard; just before onset of present illness was engaged in shovelling snow for three days; has had six children, of whom only one survives—this one has what is probably infantile palsy. (Wife seems to have had syphilis; has marked necrosis of the nasal bones.)

History of present disease. Nine weeks ago was seized with sharp general pains, more severe in right chest and in the back of the neck; also began to have fever and sweats at night. There was no shortness

of breath, no cough. Micturition was frequent and painful, and there was pain in the hypogastrium. Appetite was much diminished, but there were no gastro-intestinal disturbances. Patient lost strength rapidly. Three weeks after onset of symptoms patient fell while walking, but did not lose consciousness. On the following day the left hand and arm began to swell, and he also noticed a marked loss of power in the left upper limb; there was some pain in the left wrist. The right arm was somewhat weak.

Status præsens. A middle-aged man, exceedingly emaciated, with a skin of a peculiar bronzed hue. Tongue coated on dorsum; bright red at tip.

There is double wrist-drop; the left hand and forearm are greatly swollen; the upper arm is thin and wasted. The right hand is in a position of grasping. In both arms there is marked loss of power, and also of ability to flex and extend the hands at the wrists. Feet and legs œdematous. Knee-jerk absent; no ankle clonus. No anæsthesia; no thermo-anæsthesia.

The voice has a nasal twang; no disturbance of deglutition or of speech.

Electrical reaction. Diminished response to faradic current in left arm. The extensors of the left forearm, with the exception of the extensor carpi ulnaris, do not react at all. The muscles of the right arm respond promptly. In both forearms there is a tender spot at the point where the posterior interosseous nerve reaches the dorsal aspect of the forearm. In the thighs the reaction is good; in the legs, especially in the left soleus, it is diminished.

It was impossible to make the galvanic tests.

Lungs. Entirely negative.

Heart. A loud diastolic murmur heard best in third interspace on left side; at aortic cartilage a very faint diastolic murmur can be heard. The heart is acting violently, but is not much enlarged. The arteries are all thickened, tortuous, and atheromatous. Corrigan pulse well marked. An ulnar pulse on both sides nearly as strong as the radial.

It was concluded that the diastolic murmur heard on the left side was probably aortic in origin.

The abdomen was not distended; the organs were apparently normal, save the stomach, which was much dilated.

The temperature ran a mildly hectic course; the murmur became louder. The stools were thin and light yellow. The mental condition, at first one of hebetude, gradually passed into a mild, wandering delirium, during which the patient made many efforts to leave the bed.

The diagnosis of infectious endocarditis, with multiple neuritis, was made; typhoid fever, the other disease which the patient's condition much resembled, having been excluded.

The patient's state gradually became worse; the atrophy of the muscles increased; the thenar and hypothenar, and the interosseous muscles wasted almost visibly. The heart became feebler. Minute purpuric spots appeared on the surface of the body. The urine was generally normal; at times there was a trace of albumin; blood was never present. At no time was there tenderness in the region of the spleen.

The heart-sounds and the murmur became rapidly weaker, the impulse fluttering. Carphologia, subsultus tendinum, and stupor set in, and the patient died in a typical typhoid state.

Post-mortem. The interest of the autopsy was centred in the heart. The organ was enlarged and weighed 14 ounces; the muscle was lighter in color than normal, evidently on account of an advanced fatty degeneration. The tricuspid and pulmonary valves were normal; the mitral also, save for a slight thickening. The leaflets of the aortic valve were the seat of verrucose vegetations of unusual size. These vegetations were irregular in shape, resembling papillary growths, and were from 2 to 2½ cm. long and 2 to 3 cm. wide at their base of attachment. Vegetations also sprang from the angular spaces between the leaflets. At the junction of the anterior and posterior leaflets there was a small but deep depression, surrounded by vegetations. It appeared to be a small aneurismal dilatation.

The interior of the aorta was thickly studded with calcareous nodules and plates.

The spleen was not enlarged, but contained a large anæmic infarct 4½ cm. in width at its base. A large and a small infarct similar in character were found in the right kidney. The infarcted areas in both organs were slightly elevated above the surrounding surface, and were yellowish-white in color.

The intestines were normal. The lungs, except for some œdema, were likewise normal.

In the brain nothing pathological was discoverable with the naked eye except a small nodule springing from the right middle cerebral artery as it curved backward in the fissure of Sylvius. It did not seem to be connected with the blood-channel of the artery, *i. e.*, was not an aneurism. Its exact nature was not determined.

Sections were made from the spinal cord, the median and ulnar nerves.

May 25, 1893.

VII. TUMORS NOT OTHERWISE CLASSIFIED.

1. *Epithelioma of the perineum.*

Exhibited by Dr. James Hawley Burtenshaw.

Thomas M., aged 49 years, died at the Philadelphia Hospital, March 4, 1892. He was first admitted to the hospital during the winter of 1890–'91. The patient at that time had been a worker in a paraffin factory at Point Breeze. During the month of July, 1890, he, along with a numher of other workmen, was attacked with a peculiar skin eruption which somewhat resembled acne, but was less profuse. Upon his own body there were twelve or fifteen small-sized pustules, two of which were located upon the perineum. In the course of a week after their first appearance all had disappeared except the last named, which, probably from the irritation of the clothing, gradually increased in size. The surrounding tissues became infiltrated, and a tumor resulted, which was about the size of a walnut. At this time he was admitted into the Philadelphia Hospital. Soon afterward he was operated upon by Dr. John Deaver and the growth successfully removed. He made a good recovery and was discharged. There is no obtainable record as to whether the tumor was recognized at this time as an epithelioma or not.

In July, 1881, just one year after the appearance of the pustular eruption, while working upon a new building, he made a misstep and fell astride a rafter, the perineum being seriously injured. Almost immediately a new tumor appeared, springing from the site of the old cicatrix. He was again admitted to the hospital in November, 1891—the tumor then measuring five and one-half inches longitudinally by two or three inches transversely. The nature of the growth had been determined, and the case was reported by Dr. W. G. Porter. At his suggestion it was treated by hypodermatic injections of bichloride of mercury, gr. $\frac{1}{100}$, every six hours, the injections being made directly into the substance and around the border of the growth. This treatment was continued for one week, at the end of which time it was changed, gr. $\frac{1}{4}$ in solution being injected three times daily. Five such injections were given when, the patient becoming slightly salivated, the treatment was discontinued. At the time this was first begun there was

an ulcerating surface upon the scrotal side of the tumor one and one-half inches in diameter. Hypodermatic puncture of this area caused excessive hemorrhage. At the time this treatment with mercury was in progress the growth increased rapidly in size and the raw surface became much larger. Emaciation did not become marked until the ulceration began to increase when it became extremely rapid. Nodules were at this time to be detected upon the liver.

Complete autopsy was not permitted and, therefore, the condition of the internal organs was not satisfactorily determined. The growth was entirely confined to the skin and did not involve either the rectum, scrotum, or any of the deeper structures.

Dr. Formad regarded the specimen as typically squamous epithelioma, and remarked that few such cases have been reported, that is, not involving the rectum or scrotum.

Dr. Laplace had studied the case clinically a short time, but was unable to determine whether the liver was involved or not.

Dr. H. R. Wharton remarked that this case was of interest in connection with the study of the eruptions seen in paraffin workers. There was a case at the University Hospital of this nature, presenting ulcers principally on the legs, but also on the arms. These ulcers have much the appearance of the *warty ulcer* of Magellan, which is in reality epitheliomatous. *March* 10, 1892.

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2. *Multiple sarcoma.*

Exhibited by Dr. H. M. Fisher.

Thomas C., aged 25 years, was admitted to the Episcopal Hospital, August 25, 1891. There was a history of rheumatism six years ago, but he had had no other illness. The present illness began July 1st, with constant pain over the right side. Later there were rheumatic pains in the left hip, shoulder, and ankle, and some pain over the præcordial region. Upon admission, same pains and considerable shortness of breath were complained of. September: Tumor the size of a horse-chestnut appeared in lower right hypochondriac region, and acute pain was complained of in right shoulder, hip, leg, and ankle. November: Aspirating-needle introduced, but only a little bloody serum drawn off. Very little expansion of upper part of right lung. Over right back breath sounds feeble and distant, and fremitus much impaired close to spine.

November 28th. Two pints of bloody serum aspirated from right chest cavity.

December 1st. Numerous friction sounds heard over right chest; pericardial friction in fourth left interspace close to sternum. Some impairment of resonance under left clavicle, and profuse night sweats.

December 2d. Abdomen much distended, and sensitive. Much nervous agitation. Sweats profusely.

December 18th. Belly tympanitic, and some peritoneal effusion detected. Extreme atrophy of muscles, but muscular contractility and reflexes maintained or slightly exaggerated. Patient lies in a helpless condition, unable to move, and has involuntary evacuations. He has developed a deep bedsore over the sacrum; the urine is albuminous. He soon became weaker and more emaciated, and died December 31st.

Autopsy showed a soft, fleshy growth over eighth or ninth rib of right side in front, which, upon section, was found to be of a pinkish hue, and homogeneous in structure in its outer layers, but deeper down showed numerous lacunæ containing blood clots. The tumor had apparently originated from the rib, which was diseased, and was fractured beneath the growth.

Another fleshy and highly vascular growth was seen over the upper third of the sternum. This bone was also involved in the disease, and was fractured beneath the growth.

Numerous secondary deposits were seen in the lungs and pleura, and there was much thickening of the right pleura and numerous adhesions. The deposits in the lungs were pearly white on their surface, but pinkish on section.

In the anterior wall of the right auricle there was a growth about one and a half inches by a half inch, presenting much the same characteristics as the external growths already described. The inner surface of the auricle over this growth presented a puckered and mammilated appearance.

The aortic cusps were stiffened and thickened, the mitral leaflets also, but there were no vegetations, but two or three patches of atheroma just above the aortic valves. The liver showed very numerous growths scattered through it, presenting upon section a finely reticular appearance with numerous hemorrhages.

Microscopically. Sections of the nodules in the liver show very large areas of hemorrhage; surrounding these areas a dense fibrous network containing small lymphoid cells. There are accumulations of lymphoid cells also around the interlobular vessels, and many embryonic connective-tissue cells. The connective-tissue cells have in places undergone

mucoid degeneration, and numerous lacunæ have been formed by the destruction and disappearance of cells which have broken down and been swept away. The growths in the lungs present the ordinary appearance of a large round-celled sarcoma.

No secondary deposits were found either in the kidneys or spleen.

May 12, 1892.

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3. *Melanotic sarcoma of neck.*

Exhibited by Dr. Henry R. Wharton.

The patient from whom these tumors were removed was a young woman about 28 years of age. Some months before she came under my observation she had a small, purplish tumor removed from the neck just below the ear. When I saw her there was a well-marked swelling, occupying the infra-maxillary triangle, which was increasing rapidly in size. As the patient was seven months advanced in pregnancy, I advised that operation had best be postponed until after her confinement. I saw her again six months after this time, and found that the tumor had increased much in size, was lobulated, as though made up of several distinct masses.

The patient was etherized and the tumors were exposed and removed, and consisted of masses such as I show here. The patient did well, and for three months I lost sight of her, but at that expiration of this time I saw her again, and she had a small growth about the size of a walnut behind the site of the cicatrix of the previous operation. The growth is probably a melanotic sarcoma, which is a very malignant form of sarcoma.

Dr. Stengel called attention to the seeming influence of pregnancy in stimulating the growth to activity, and cited a case recently under his own observation, bearing out the same idea. A woman who had had sharp pains in the breast and a hard nodule to which no attention was paid during its existence of several months or more, became pregnant. It was noted that the abdominal swelling during pregnancy was considerable, but this excited no particular comment. After delivery the abdomen remained persistently large, and it was discovered that the woman was suffering from secondary cancer of the liver. The woman, a lady of intelligence, asserts positively that there was no abdominal swelling prior to the pregnancy.

In view of cases of this class, the speaker asked if it would not be the part of wisdom to operate in operable cases as early during pregnancy as possible.

Dr. Allyn also referred to a case in which during pregnancy a dark mole or nævus of the neck rapidly grew and became a fungating melanotic sarcoma. The speaker further alluded to the difficulty in distinguishing between such moles and true nævi. *December* 8, 1892.

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4. *Lipoma with calcareous infiltration.*

Exhibited by Dr. H. F. Slifer.

This specimen was removed from a man aged 50 years. The man was a blacksmith and in good general health. Fifteen years since he received a heavy blow on the leg, and a year subsequently noted the appearance of a lump, which gradually increased in size until it reached the diameter of an inch and a half. At this point the growth remained stationary until five years since, when it again began to grow rapidly until it reached its present size of about four inches in diameter. Two months since the tumor became irritated by friction and began to suppurate, discharging offensive material. This has reduced the size somewhat, so that in reality the growth was larger than at present. On removal it was found to be a lipoma with extensive calcareous deposit, rendering the growth almost stony hard. The extent of this infiltration has seemed to me unusual, though I am aware of the occurrence in some cases.

Dr. Coplin recalled a case in which Dr. Gross removed thirteen partly calcified lipomata from a case in which multiple growths were found in various situations. *March* 23, 1892.

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5. *Retro-peritoneal sarcoma.*

Exhibited by Dr. W. E. Hughes.

Patient, a miner, had been healthy until within two weeks of the time he came under observation. At that time he noticed, while lifting a barrel of sugar, that something gave way. Following that he had pain in the belly, nausea, vomiting, looseness of bowels, rapid loss of strength. When first seen he was very anæmic and cyanotic. Marked

distention of the external jugular and abdominal veins. A hard, irregular, nodular mass was felt in the umbilical and left lumbar regions. The mass was not affected by respiratory movements.

Post-mortem examination revealed a mass, apparently developing in the retro-peritoneal lymph glands, as large as a head. Many secondary growths in the mesenteric glands; and the duodenum, head of pancreas, kidneys, and heart muscles were very much involved. Tail of pancreas free. The disease seemed to be sarcoma with multiple secondary foci.

Previous to death an examination of the sputum revealed many tubercle bacilli. The lungs, post-mortem, revealed but one small doubtful nodule.

Dr. Meigs spoke of the secondary involvement of the eye by metastasis.

Dr. Hughes adds that the patient had a profuse hemorrhage shortly before death, and this had led to the examination of the sputum. The question of the dissemination of sarcoma is most interesting, and in the present case might lead to profitable study. *May* 11, 1893.

VIII. MISCELLANEOUS.

1. *Lesions secondary to pulmonary tuberculosis.*

A discussion led, at the invitation of the Society, by Drs. John Guitéras and John H. Musser.

Secondary tuberculous lesions of the nervous system.

Read by Dr. John Guitéras.

Dr. Guitéras opened the discussion by a consideration of tuberculous lesions of the nervous system. The brain lesions may be considered from the clinical and anatomical standpoint as of two kinds: (*a*) tubercular meningitis and (*b*) solitary tubercles. The first occurs especially in the practice of the general practitioncr, the second in that of the neurologist.

When we come now to the study of tubercular meningitis, we shall find this composed of two component parts, the tubercle and the accompanying inflammation. The tubercles are of the miliary variety, and especially abundant toward the base of the brain. They are most numerous along the bloodvessels, or rather, perhaps, along the lymphatics which accompany the bloodvessels. As the case progresses the tubercles undergo certain changes, becoming more and more caseous, but it is to be remarked that caseation is a less prominent feature in tubercles about the meninges than elsewhere. Along with the occurrence and growth of the tubercle there is seen congestion of the membranes, then exudation of serum fibrin and leukocytes, and finally, from excessive numbers of the latter, a true purulent collection. The tubercles, though most prominent in the meninges, do to a certain extent penetrate the cortical layer of the brain substance, and by extension along the choroid plexus they also reach the ventricles and set up internal hydrocephalus, with distention of the cavities. Histologically, there is nothing new or distinctive about the tubercle of the brain, but not infrequently it shows in an exquisite manner the manifestations described as characteristic of tubercles. We see very well the process of formation of the epitheliodal cell, with the infiltration of round cells surrounding them, and in the centre of the mass the gradual loss of chromophilic properties as coagu-

lation necrosis advances. The giant cells are present, as in other localities. The inflammatory disease of the meninges accompanying the growth of tubercle is a distinct acute process, often manifesting itself very like a simple meningitis, but in other cases ill-defined, coarse, and very difficult to diagnose.

The solitary lesion of tuberculosis is quite different from the above. In this case there is no general serous inflammation and no tendency to extension of the process. It is a thoroughly localized disease, in which one tubercle after another is added until a mass or tumor results. As this increases in size, the oldest tubercles in the centre begin to degenerate and become cheesy, the outer zones remaining as a rosy-gray layer containing translucent miliary tubercles. Occasionally the peripheral nodules show tendency to active formation of fibrous tissue, and the tumor tends to be encapsulated.

There is nothing new to say regarding the lesions of tuberculosis. As far as this part of the question is concerned, the last word was said long ago, and even the immense light thrown on the study of tuberculosis during the past year has added no material contribution to our knowledge of the anatomy or histology. We seem to have reached a point beyond which our instruments of precision are not powerful enough to carry us, and the histology of tuberculosis, as known to-day, is essentially the same as that of twenty years ago. We cannot hope, then, to enter upon any discussion of this part of the subject.

All that has been added of recent years, and all that promises in any way to lead to a cure, is concerned with the etiology of the disease. The experiments of Villemin paved the way for the later researches of Koch and the discovery of the bacillus. Villemin was the first to demonstrate beyond doubt the infectious nature of the disease, and Koch succeeded in isolating the infectious agent.

When we come to the consideration of cerebral tuberculosis alone we shall find some difficulty in explaining the channels of infection in certain cases, but it is in this very line that recent knowledge has given us light. Cerebral tuberculosis—both the meningeal form and the solitary tubercular tumor—are more common in childhood than in later life. Tubercular meningitis is, in the vast majority of cases, secondary, though in many of the older reports no primary lesion is described. With our better knowledge of the primary lesions, however, it is now almost, if not invariable to find a primary focus of disease. The consideration of the lines by which infection spreads is of great interest, but often not a little obscure. To take a few of the simpler cases: the meninges may become involved by direct infection from disease of the cranial bones, as

very frequently the temporal, or of the mucous membranes of the various cavities, as the ears, nose, etc. It is probable that infection from the nasal mucosa is much more common than we have been accustomed to think. The chronic catarrhs of scrofulous children are doubtless quite frequently really tubercular in nature, and it is easy to see how infection of the meninges might occur. The lymphatics of the nose may be injected from the large subdural space, and we see, therefore, the possibilities of this channel for infection. The study of hæmatogeneous tuberculosis of the brain, that is, cases in which the infective agent has been carried by the bloodvessels, is more difficult than that of the previously-mentioned cases; for we are met at once by the fact that the lesions do not occur in the final distribution of the vessels, deep in the gray or superficially in the white matter of the cortex, where embolic manifestations ought to occur, but in the meninges or superficial layers of the cortex. We may possibly find the proper explanation of this in the fact that the brain substance is an unfavorable soil for tubercular new-growth, and that the bacillus deposited there through the bloodstream is carried outward again by the lymph paths to find a lodgment and further multiplication in the meninges. Probability is lent to this view by the peculiarly well adapted arrangement of the lymphatics, which run by the side of the veins and toward the base, and it is to be noted that the latter is the favorite seat of tuberculous disease. In this connection it is interesting to consider Prudden's recent experiments. Injecting dead tubercle bacilli into the general circulation Prudden was able to produce lesions in liver, lungs, spleen, etc., but none in the brain. The reason of this may lie in the activity of the lymphatic circulation. The bacilli readily escape from the bloodvessels and are probably at once removed by the lymphatics, having had no opportunity to produce lesions. In the other organs dead tubercle bacilli produced changes, to a certain extent, strikingly like those of tuberculosis, but differing in several essential features. Injection of living bacilli produce the disease in all of its features, but introduction of the dead germ, whatever local lesions it may produce, cannot and do not give rise to an infectious disease, with possibility of further spread. Another essential difference is found in the subsequent changes which the pseudo-tubercular lesions undergo. One of the ruling characteristics of tuberculosis is the failure of proper blood-supply, and therefore, sooner or later, the occurrence of degenerative changes. With the lesions produced by the dead bacilli there is an entirely different story. In this case we have exactly the behavior that is seen on introduction of any other body having positive chemo-lactic properties: round cell infiltration, formation of new cells

and giant cells, but also formation of new bloodvessels to supply nutrition to the proliferating structures, and therefore no degenerative changes. A further reason for degeneration in tuberculosis is the specific action of the bacillus itself, which tends to incite coagulation necrosis.

Secondary tuberculous lesions of the gastro-intestinal tract.

Dr. Musser followed with a consideration of the lesions of the gastro-intestinal tract, saying: It is often difficult to trace the sequence of involvement in the different organs when a number have been affected, and it is sometimes difficult to decide upon the seat of primary lesion. Still it has been possible to collect certain statistical evidence of the frequency of gastro-intestinal tuberculosis. Woodhead has made an elaborate tabulation, and I have myself recorded the results of 93 autopsies in which careful examination of the various organs was made. Of these cases, in 70 there was primary lesion of the lungs, and in 63 of these 70 secondary involvement of the gastro-intestinal tract.

The tongue is a somewhat common seat of tubercular lesions, but I have never seen such secondary to pulmonary tubercle. On the contrary, there is usually a localized ulceration which is primary in the locality. I have seen but one instance of tuberculosis of the tonsil: this in a young man 18 years of age. The tonsils were enlarged, pale and honeycombed by ulcers which showed tubercles at the base and gave rise to copious discharge of purulent material. Tubercle bacilli were found in the discharges and in the tonsils on histological examination.

The œsophagus has never been the seat of tubercular disease in my experience. The stomach is an infrequent seat of tuberculosis, and but one case has come under my observation. The only other case recorded in Philadelphia was that reported by Dr. F. P. Henry. From a critical study of the literature of the subject I find but twenty or twenty-five cases.

The lesions are generally multiple and may be as small as a split pea. Usually, however, the ulcers are large and are most frequently found on the lesser curvature. The edges are indurated from collections of cheesy material in the tissues, and the bottom of the ulcer is studded with tubercles. The peritoneal surface opposite the ulceration is always the seat of a localized inflammation. Clinically, many cases are characterized by hemorrhage from the stomach, and this may be the first indication of the disease which otherwise remains latent in a certain number of cases. When hemorrhage does not occur in these cases, perforation

of the stomach may be the first sign of the process. The *post-mortem* appearances readily distinguish tubercular ulcers from ordinary round ulcers or cancer, but the most careful examination should be made to decide positively. It is interesting to study the etiology of this condition. In all probability there must first be some localized abrasion or erosion of the epithelium before the bacilli can find a nidus for development. The latter are usually derived from the lungs, the patient swallowing sputum. This was a marked feature in the case which I have observed. There are certain conditions, however, which make it difficult for the bacillus to find a resting-place in the stomach. The acidity of the gastric juice is highly unsuitable to their growth, and the mobility of the organ and the rapidity with which the contents are hurried along further contribute to the difficulty in the way of gastric infection.

Intestinal tuberculosis, on the other hand, is a common accident in pulmonary tuberculosis. Here the conditions are highly favorable ; the juices are alkaline, the gut is more or less fixed in position, the contents are not hurried along so rapidly, and the lymphatic supply is particularly rich, which favors the formation of lesions in disseminated lymphogenous tuberculosis. More commonly than that form, however, is that in which swallowing of the tubercle bacillus in food or sputum leads to the intestinal ulceration. The seat of ulceration is most frequently the lower part of the ileum, and secondarily it occurs also in the large gut. The vermiform appendix is often involved, and occasionally the disease begins as an attack of appendicitis; two cases of this kind have come under my observation. Without previous diarrhœa the disease begins with sharp pain in the right iliac fossa, fever and other symptoms of appendicitis. The ulcers in the intestine may be small, involving only the solitary follicles, or large when Peyer's patches and the surrounding tissues are necrosed. They are readily distinguished from typhoid ulcerations by their greater number, by their transverse direction and by the miliary tubercles seen on the serous coat.

I have never seen tubercular ulcerations of the rectum, except as we see it in peri-rectal abscess and fistula-in-ano.

The liver is a frequent seat of secondary tuberculosis. The lesions are nearly always small, and may require a magnifying glass to bring them into view. Rarely ever do we find large cheesy masses; I have never seen such. The spleen likewise is involved in general tuberculosis, as also the peritoneum and the lymph glands of the abdomen, but time does not permit discussion of these points.

Dr. Flick did not agree with those who regard intestinal tuberculosis

as always secondary to lesions in the lungs, though this is the rule. In infancy he thought that tuberculosis of the intestines and brain is a much more common condition than has generally been believed, and these cases are always or usually primary. The disease called marasmus is probably always tuberculosis of the intestines or lymphatic structures of the abdomen. Convulsions of children, also, are not rarely a manifestation of tubercular disease.

Modern surgery has pointed out the frequent occurrence of tuberculosis where it has not been suspected, as in operations on the abdominal cavity disclosing latent tuberculous lesions. In one case he saw pulmonary tuberculosis develop after a primary lesion in the pelvis, and he was disposed to regard the saying among people, that girls who have evidence of pelvic trouble will have tuberculosis as a result, as carrying more than a grain of truth.

Dr. Musser agreed entirely with Dr. Flick in his belief in primary tuberculosis of the gastro-intestinal tract, but limited his remarks to the secondary forms in accordance with the scope of the discussion.

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2. *Visceral syphilis.*

Exhibited by Dr. Allen J. Smith.

The person from whom these specimens were obtained, C. M., was a man whose life prior to his admission in the University Hospital in the the private service of Dr. J. William White, had been one of greater or less, but continuous dissipation. He was about 45 or 50 years of age, married, but childless. Admitted venereal disease, gonorrhœa, and syphilis.

The specimens, as presented, consist of the entire urinary and genital apparatus, removed, at the request of the attending surgeon, *en masse.* The right kidney is enlarged to half again the usual size; its capsule is somewhat thickened and does not pare off from the surface with much ease. There are a few small subcapsular cysts. The cortex is decidedly thicker than normal, and the entire organ is congested. In contrast to the right, the left kidney, distorted by very marked scars, is diminished to less than half the ordinary size of a kidney. Its capsule is decidedly thickened and adherent, and the organ, on section, shows marked disfiguration by cicatrices and by an interstitial nephritis. The cortex is quite thin; the pelvis of the right organ is dilated; the ureter distended

and its walls thickened. The pelvis and ureter of the opposite organ are not markedly altered. The bladder wall is thickened, the mucous surface is rugous and covered with mucous, and here and there are patches of pigmenting deposit. The middle lobe of the prostate is somewhat enlarged; the urethra is the seat of several fibrous strictures. The glans penis shows the scar of an old sore; and this, the history and the peculiar one-sided atrophic change of the kidney may be regarded as of interdependence. A number of specimens, shown in this Society from time to time, present, if I recall aright, the same distinctions of visceral syphilitic atrophies and corroborate what has been regarded as a more or less definite rule, that syphilitic changes from gummatous formations and degeneration are usually unilateral lesions. *September* 10, 1891.

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3. *Specimens from a case of tuberculosis treated by Koch's method.*

Exhibited by Dr. Allen J. Smith.

The patient from whose body these specimens were removed was one of a number who served as subjects for the University of Pennsylvania's Koch Commission some months ago. The patient, C. L., was a male of about 28 or 30 years of age, of a tubercular family, and with tuberculosis of several years' duration at the time of his admission. His death occurred several weeks after admission to the hospital, a temporary improvement in his symptoms, after undergoing injection with tuberculin, being soon followed by a serious and eventually fatal decline.

At the autopsy widespread tuberculosis was found, involving the lungs, pleura, intestines, and kidneys. The lungs, which are before the Society, are decidedly infiltrated with tuberculous matter, and in each apex is to be seen a considerable cavity. Besides these larger cavities a number of smaller ones may be found in the upper and middle lobes and in the lower lobe of the left side. The point of especial interest in the specimen is the degree of organization which may be noticed in the walls of all these cavities, a pink line of recently formed connective tissue, about one and a half lines in thickness, extending completely about all of these cavities. Similar pink zones may be found, moreover, about the larger tubercular masses which have not as yet broken down. The walls of the cavities are, moreover, extremely clear on their inner surface, and the wall held between the thumb and finger is of leathery consistence. These same peculiarities, due apparently to efforts at organization of

tissue about the diseased areas and their isolation from the rest of the organism, may be even more easily observed in case of the intestinal lesions. The entire length of the small intestine, it will be noticed, is the seat of numerous tubercular ulcers of the general characteristics of such lesions, especially low down in the ileum, but in many cases showing a cicatrization, which must indicate the action of an actual healing process. The bases of many of these ulcers are firm and leathery, thoroughly organized, the edges almost on a level with the surrounding mucous surface, and, save for their slightly pinkish line of the new tissue, they would be regarded as complete in their healing. Lower down the irregular overhanging edges of the ulcers become more marked, although in all cases there is a very evident effort at repair; and in none of the lesions can the ordinarily necrotic changes be seen to be anything like the same degree as in an ordinary case.

Unfortunately, the exhibitor has thus far had no opportunity to make a microscopic examination of the structures, which in their macroscopic appearance indicate such satisfactory results from the employment of this mode of treatment. The patient furnishing these specimens was the only one who died in the University Hospital, and no further opportunity has therefore been afforded to witness the effect of the treatment.

It may be added at this time that as microscopist to the Commission above referred to, the speaker had occasion to examine more than six hundred preparations of sputum obtained from some thirty individuals. In spite of the criticisms which have been made against the use of this new therapeutic agent, this experience has forced the belief in its possession of a modifying influence, not only from the evidence of the specimens exhibited here this evening, but also from changes in the appearance of the sputum. As a rule, the sputum, after treatment by tuberculin, becomes of a thinner consistence, loses its thick, glairy character, and becomes lighter in color. Under the microscope the tubercle bacilli, found in every instance, may in a general way be said to have diminished somewhat in number after the first few days of treatment; but in no instance did they disappear or even decrease very greatly. The bacilli, however, did not, after some days of treatment, show as marked sporulation as at first, and small, misshapen, and large branched forms of the germ were quite common in the later specimens, as a rule. These alterations indicate the operation of some influence, which may not necessarily be an evil one, as is coming to be the general expression in relation to tuberculin. The development of branched forms is indicative, it seems probable, of a change of the bacillus to the morphology of those fungi which are characteristically found in localized processes.

So the lesions in the case of C. F. appear localized to a marked degree; and it seems not unlikely that it will eventually be found that in early and picked cases the use of tuberculin will tend to localize and isolate tubercular processes, which ordinarily would become general and fatal.

September 10, 1891.

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4. *Specimen of skin; suspected leprosy.*

Exhibited by Dr. H. M. Fischer.

These portions of skin were taken from the buttocks of a Chinaman, who recently died in the Pennsylvania Hospital with symptoms of spinal meningitis. The day before death nodules were found in the skin in several places, with discoloration over them. The condition of the patient made it impossible to determine whether there was anæsthesia or not. The case was considered as one of leprosy. Microscopic sections showed increase of connective tissue about the sweat-glands but no definite lesions.

October 22, 1891.

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5. *Superficial lesions of gout.*

Exhibited by Dr. J. H. Musser.

I wish to present some paintings made for me by Dr. Taylor, which illustrate external lesions of gout. The first shows the hands and feet of a woman suffering with gout, and who also has an old cardiac lesion dating from childhood, and undoubtedly of rheumatic origin. There are nodules over the tendo Achillis, and some also about the hands, under the skin and freely movable.

The next illustration shows nodules on the hand, from a case of gout, specimens of which were exhibited to the Society by Dr. Pearce, of the Presbyterian Hospital, on September 10th. This man had advanced gouty nephritis.

These nodules of gout are comparatively rare in this country, but rather common in England. A marked case is reported of a man who had such marked chalky nodules that the skin was perforated and he could keep his score at cards by writing with his knuckles.

October 22, 1891.

6. *Congenital cystic elephantiasis. A further report of a case of multiple tumors in the newborn.*

Read by Dr. W. Reynolds Wilson.

In January last I had the pleasure of exhibiting to the Society the specimen of a fœtus born at the end of the eighth month, and presenting an unusual pathological condition.

The specimen measures forty centimetres in length and weighs five and one-half pounds. A large, fluctuating tumor extending from the

axilla on the right side, to the crest of the ilium, increases the circumference of the chest to thirty-eight and five-tenths centimetres. Similar tumors of varying consistence cover the extensor surfaces of the arms and the dorsal surfaces of the fingers and toes. Both thighs are enlarged, evidently by a hyperplasia of the subcutaneous connective tissue. It will not be necessary to go into a more detailed description of the macroscopic appearance of the specimen, as the attention of the Society

has already been called to this point, and as the photograph at hand illustrates the condition.[1] When I reported the case to the Society, at a previous meeting, I was led to conclude from the appearance of the placenta and from sections from the various tumors that we were dealing with a myxomatous condition. Although the consistence of some of the tumors, as well as the character of some of the tumor constituents, seemed to warrant this opinion, on more careful examination it was apparent that the existence of the tumors depended upon a dilatation of the lymphatics.

The literature upon the subject of lymphatic tumors revealed the fact that the case with which we have to deal belongs to an unusual variety. The microscopic examination showed the existence not only of lymphatic enlargement but of hyperplasia of connective tissue. Sections from the following tissues were examined: from the placenta, the cord, the skin and subcutaneous tissue of the thigh, from the wall of the large cyst of the thoracic region and from one of the tumors of the hand. The placenta macroscopically presented no pathological changes. It was friable but normal in size, thickness and color. The microscope shows, however, in some places, an increase in the ground substance of the villi. The decidual cells appear to have lost their outline and to have undergone a granular degeneration. These changes produce the structureless appearance which in a more advanced state is characteristic of myxomatous placentæ. There are, however, no cysts, and the pathological changes are of no importance, considered either in respect to their character or extent. That is to say, those lesions in the placenta to which we should be inclined to attribute any interruption of development in the fœtal tissues are absent. Local lesions in the maternal tissues, namely, pathological changes in the uterine decidua, affecting the placenta, are also absent. For instance, the placenta shows no inflammatory infiltration due to irritative changes. The mother toward the end of her pregnancy suffered from hydramnios. In view of this fact there is another possible lesion of the placenta which should be looked for in our study of the case. That is, the existence of changes indicative of kidney disease. While infarcts of nephritic origin, due to necrosis of the villi from changes in the vessel walls, were absent, although here and there areas of dense fibrous tissue in the neighborhood of the larger vessels are apparent. Whether the lesions to which reference has been made are of sufficient importance to be considered in connection with the changes in the fœtus is an open question.

[1] The cut is taken from the American Journal of Obstetrics and Diseases of Women and Children, vol. xxiv., No. 10.

The section of the tumor of the hand represents the constituents of the various tumors, although macroscopically there is a difference between the tumors of the extremities and those on the trunk. The latter are composed of large cysts with thin walls, while the former contain dilated lymph spaces with thickened walls and hyperplastic connective tissue. These spaces are lined by proliferated endothelium and cylindrical cells, and in some instances they are filled with broken-down lymph corpuscles. Throughout the section areas appear in which the fibres of connective tissue are replaced by a homogeneous, granular tissue, beset with nuclei in a state of granular degeneration. There are no branched cells indicative of myxomatous tissue of mature growth. Œdema is present. The section from the thigh indicates more fully the true nature of the condition. We have a hyperplasia of connective tissue with an increase in the number of bloodvessels, their neighborhood being marked by an infiltration of small cells. The lymph spaces are dilated, but are apparently not in communication. The increase in connective tissue is confined to the subcutaneous tissue. This hyperplastic growth, together with the thickening of the corium, have led to an increase in the size of both thighs. The reddish discoloration of the lower extremities is due to dilatation of the bloodvessels.

The contents of the cysts which compose the larger tumors is a serous fluid, in some instances mixed with blood. It contains here and there large, transparent, faintly granular cells, with multiple, vesicular nuclei. These are probably endothelial cells in a state of mucous degeneration. The fœtus showed no ascites and no abnormality of the internal organs. The section from the cord reveals dense infiltration around the vessels, with thickening of the media and adventitia of the arteries.

In order to study the pathology of this condition we must observe the changes in all of the tissues under examination, and endeavor to detect the lesion which is common to all. In examining the fœtal tissues with this end in view, we find a common element in the pathology, namely, a dilatation of the lymph spaces. Although this dilatation is found in all of the tissues, the accompanying changes are different in different sections. For instance, in the section from the thoracic tumor the dilatation is so extensive as to give rise to large, thinned-walled cysts, while in the section from the hand the cysts are smaller and are bounded by the hypertrophied lymphatic walls, and in the section from the thigh the dilatation is only moderate and the spaces are bounded by hyperplastic connective tissue. Another general characteristic of these neoplasms is the fact that they are confined to the skin and subcutaneous connective tissue. There is no obstruction in the line of the larger

lymphatic trunks and thoracic duct. We have then to do, first, with either a general dilatation of the subcutaneous lymphatics with stagnation of lymph from obstruction, or with a neoplastic growth from an increase both in the number and in the volume of the lymph spaces; and secondly, with a hyperplasia of connective tissue more or less common to all the tissues, with increase in the number of vessels and thickening of the skin. From these facts we are warranted in making the following diagnosis: "Telangiectatic elephantiasis."

After a careful study of the literature upon this condition in the newborn, I have determined to adopt the title of "congenital cystic elephantiasis."

The case was first reported to the Society as an instance of multiple cystic myxomata. This diagnosis was made from the appearance of the beginning myxomatous degeneration of the placenta and from the areas in the fœtal tissue which resembled myxomatous tissue of immature growth. The semi-fluctuating tumor of the thorax was not cut into at first, because it was deemed advisable to preserve the specimen intact. When this tumor was examined, however, it was found that the jelly-like consistence had been due to the colloid material contained within the cysts, and not to an accumulation of embryonic tissue, forming the ground substance of the tumor, as has been supposed.

In consideration of the fact that the increase of tissue is confined to the subcutaneous layer, it was natural to conclude that we had to do with a process by which the tissue normally present in this region, namely fat, was being replaced by new tissue. In fact, an examination by the microscope showed that this was the case; for the fatty tissue in some of the sections is absent. In others, however, notably those from the thigh, it exists in normal proportions. According to Virchow, the development of fatty tissue in the embryo is preceded by a deposit of embryonic tissue. Should the latter from some cause fail to undergo the normal metamorphosis, and at the same time take on a hyperplastic growth, we should have, in time, the development of mature myxomatous tissue.

The increase, however, in connective-tissue elements and the dilatation of the lymphatics appear on closer investigation to bear such an important relation to the development of the tumors that it has seemed wise to abandon the former diagnosis.

There are various hypotheses as to the etiology of this condition. Busey advocated the theory of obstruction with dilatation of the lymph-channels and secondary hyperplasia of the connective tissue. It has been demonstrated, however, that obstruction of the lymphatic system

from pressure will not produce a condition of lymphatic ectasis as long as anastomosis exists and the quality of the blood remains normal. Moreover, the hypertrophy of the lymphatic walls and thickening of the cyst walls cannot be considered as the result of stasis alone, nor, on the other hand, as the result of a primary inflammation of the surrounding subcutaneous tissue. Nasse cites, in opposition to this view, the fact that cicatricial tissue, in which by contraction of the connective-tissue fibres the lymph spaces become dilated and distorted, offers through the stagnation of lymph no predisposition to the growth of lymphangiomata. Friedburg suggests that this condition may have originated either in some affection of the vasomotor nerves, or from an impediment of the circulation in the lymph-vessels, produced by a swollen lymph-gland or other tumor; or in a morbid condition of the blood with consequent infiltration of the tissue and proliferation; or from inflammation of the skin and subcutaneous connective tissue, lymph-glands, and vessels.

In a case reported by Neelsen the evidences of obstruction were absent, and inflammatory changes (infiltration of connective tissue with increase in the number of vessels) existed in a subordinate degree, although the fœtus presented cysts as large and as numerous as those of the specimen in the present case. Török inclines toward the opinion of Wegner, that the tumors are of neoplastic origin formed by new lymphatic branches developing from angioblasts, as in the case of angiomata, and dilating to form cysts. In support of this theory he adduces the following facts: First, that the lymphangiomata frequently find their origin in the subcutaneous fat tissue, which is naturally poor in lymph vessels, and secondly, that the rapid growth of these tumors, where their origin cannot be ascribed to obstruction, can only be explained by the growth of new vessels.

In the further study of the etiology of such cases of congenital affection, we cannot do better than refer to the paper of Neelsen quoted above, in which he adopts a theory entertained by Klebs and Schulze—that is, that the final pathological changes, namely, increase of connective tissue, dilatation of lymph-spaces and hypertrophy of lymphatic walls, are due to a chronic inflammatory œdema. In cases of hydræmia, where some chronic blood dyscrasia exists, as in syphilis, the quality of the blood, effecting an alteration in the vessel walls, is the cause of the transudation. In the case reported by Neelsen, the mother suffered from nephritis and anasarca during the later months of pregnancy. The fœtus, however, as in my case, exhibited no hydrothorax or ascites; in both cases the lymphatic system of the subcutaneous tissue was alone affected. Neelsen concludes with Cohnheim that the œdema occurs

only in those tissues where the vessels are in a condition to favor transudation, a condition similar to that existing in the early stage of inflammation—in his case in the bones and subcutaneous connective tissue.

The cause of hydramnios in the present case remains unexplained, inasmuch as the source of the fluid is to be found neither in obstruction to the blood stream between the fœtal and maternal circulations, nor in changes in the placental circulation sufficient to account for the excessive secretion, nor in the fœtal circulation, causing hypertrophy of the heart and over-activity of the kidneys, nor in the condition of the infant's skin. The earlier children in Neelsen's cases suffered from tubercular affections of the bones and joints, and from skin diseases incident to the same cachexia. The mother had also exhibited tubercular tendencies. This fact and the existence of nephritis account for changes in the maternal blood sufficient to produce the œdema, and the consequent irritation of the skin and subcutaneous tissue, with hyperplasia. We have no such history in the case which has been reported, and although we may look to the theory of blood dyscrasia, with œdema and secondary inflammatory changes of the subcutaneous tissue, as the most plausible, the etiology is still obscure. Nor does the absence of irritative or marked degenerative changes in the placenta contribute to lessen this obscurity.

Dr. Formad: There are cases on record of multiple myxomata in the newborn, and I should prefer to consider this case as such rather than as telangiectatic lymphangioma. There is certainly in these specimens evidence of myxomatous degeneration and hyperplastic growth, and in elephantiasis or lymphangioma such does not occur. There are here many of the features of a condition of myxœdema.

November 12, 1891.

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7. *Anthracosis.*

Exhibited by Dr. D. J. M. Miller.

I. C., male, aged 57 years, a miner, was perfectly well until two years ago, when he took a severe cold. Since that time he has had constant cough, shortness of breath, and dark-colored, muco-purulent expectoration. These symptoms have improved from time to time, but he has never been entirely free from them. His family history has been remarkably good. On February 2, 1892, he was admitted to the Episcopal Hospital for the third time, having been in the institution twice

during 1891 for the same trouble, leaving, after a few weeks' stay, very much better. At the date of his last admission he presented a pale and somewhat emaciated appearance. His finger-nails were slightly clubbed, his appetite poor. He complained principally, however, of shortness of breath and of a slight cough that troubled him mostly in the morning, and which was accompanied by a small amount of grayish, muco-purulent sputa. He seemed very weak. Physical examination revealed the signs of chronic bronchitis and emphysema; chest everywhere tympanitic, prolonged, wheezy expectoration, and râles of various sorts—creaking, mucous, sonorous, and sibilant. The cardiac sounds were weak, but no murmur was heard. The apex was displaced to the left. The hepatic area seemed normal; the spleen enlarged. No albumin was found in his urine. The patient at first improved, but soon began to fail again. On March 9th he complained of severe headache and weakness, and his feet had begun to swell. On the 13th he seemed nervous and depressed, and still complained of headache. The cardiac sounds and pulse were very feeble. His weakness and headache continued until his death, which occurred rather suddenly early on the morning of the 14th.

Autopsy. Both pleural sacs were completely obliterated.

Right lung was jet-black in color throughout. On section an ink-like juice flowed from the cut surface. The lung substance was tough and fibrous, cutting with difficulty. This condition was present in all portions of the lung, but was particularly marked at the apex, which was so adherent through its pleura to the chest-wall that it was torn in removal. The left lung was in a similar condition at the apex; throughout the remainder the change existed in the form of numerous areas of black fibrous tissue. The bronchial glands were enlarged and jet-black on section. The bronchi themselves did not share the pigmentation. The pericardial sac contained five ounces of serum.

The heart was enlarged; both ventricles hypertrophied and dilated. There were small calcareous deposits on the mitral leaflet.

The liver was small; the left lobe almost entirely absent; its cut surface yellow in color and moderately fibrous in character.

The spleen was congested and enlarged, but contained no pigment.

The kidneys were enlarged; the cortical portion seemed relatively small when compared with the medulla; the surface nodular and capsule adherent.

The brain itself seemed normal. The dura mater was closely adherent to the inner table of the skull. There were several patches of imperfectly organized lymph about the vertex near the longitudinal fissure

between the brain and pia mater. Death was probably due to gradual cardiac and circulatory failure. It is interesting to note that the bronchi were free from pigmentation, which was probably due to the inability of the pigment to penetrate the basement epithelium lining those tubes.

For making the autopsy, as well as for the notes of this case, I am indebted to Dr. W. W. Ashhurst, the resident physician.

April 14, 1892.

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8. *The factors concerned in the production of suppuration.*

Read by Dr. A. C. Abbott, at the Semi-annual Meeting, April 28, 1892.

That suppurative processes occurring spontaneously in man are in one way or another dependent upon the presence of bacteria in the tissues is a fact too well established to require discussion before this assembly.

Since the observations of Rindfleisch,[1] Waldeyer,[2] Von Recklinghausen,[3] Klebs,[4] and others, upon the suppurative diseases of wounds, which pointed in the main to their being of an infectious nature, our knowedge upon the subject has made such advances that we are to-day in a position to understand and control the more important of the hitherto obscure conditions underlying these processes. In asking your attention to a consideration of the factors concerned in the production of suppuration, it is with a view of discussing some of the more recent, and in certain instances not yet proved, hypotheses that have been advanced in explanation of the phenomenon.

Since the work of Ogston,[5] Rosenbach,[6] and Passet,[7] opinions that have been held in regard to suppurative processes in general have, in the main, been in accord with evidence accruing from bacteriological studies; and have agreed in considering the organisms described by these observers as the specific etiological factors in the production of the conditions. But in the light of more recent observations, to which I ask your attention in this paper, it appears that we shall have to subject these views to a certain degree of modification, and consider suppurative processes as a possible result under peculiar circumstances, not only of organisms other than those to which specific pyogenic properties have been attributed, but also to chemical substances that in some instances are, and in others are not, connected with bacteria or their life processes.

So interesting are the phases through which our acquaintance with

this phenomenon has passed in its evolution to the position it now occupies that, at the risk of presenting observations already known to many of you, I shall ask your attention to a superficial review of some of the more prominent contributions upon which our present knowledge of the subject is based.

Up to the date of its publication, the most important observation that was made upon the bacteria found in the pus of abscesses in human beings were those of Ogston,[5] 1881. In this publication he called attention to the constant presence in pus of spherical organisms that were seen to have a characteristic grouping. He noticed that these organisms were never absent from the pus of acute abscesses, and that, on the contrary, they were rarely to be seen in the contents of cold abscesses. He found that in the acute, circumscribed abscess-formations, the organisms most commonly present were spherical, and were grouped together in irregular clusters, somewhat resembling clusters of grapes. On the other hand, in the spreading or phlegmonous suppurations, the organisms most commonly present were not found in these irregular clusters, but rather in chains somewhat resembling strands of beads. For the former, the clustered cocci, he suggested the name staphylococci, and for those appearing in chains he proposed the name streptococci.

From his observations he concluded that the staphylococci were most commonly concerned in circumscribed suppurations which were characterized by necrosis about the point at which the organisms were located, while the streptococci, in his opinion, gave rise to tissue-changes, most conspicuous in alterations in the lymphatics, and consequently in their spreading tendencies.

As a result of injections into lower animals of material containing the staphylococci a condition was produced that resembled somewhat a soft chancre. "The centre of the mass," he states, "was of a yellow color, and was soft and necrotic in character. It contained a mass of closely-packed staphylococci, about which was a zone of tissue of a waxy homogeneous appearance, that increased in extent as the cocci continued to multiply." The changes in the tissues resulting from the introduction of the streptococci were different from this. In general, there was a waxy necrotic appearance of the tissues about the point at which the streptococci were deposited, but the further behavior of the organisms differed markedly from that of the staphylococci. Instead of a localization at the point at which they found access to the tissues, there was a tendency toward an extension along the lymphatic spaces, and the ultimate production of a process analagous pathologically to erysipelatous inflammation.

In 1884, Rosenbach[6] confirmed the observations of Ogston, and, by

means of the methods recommended by Koch,[8] succeeded in isolating in cultures the organisms that Ogston had described from microscopical examination. He found, as Koch had already suggested, that the organisms commonly concerned in suppurative inflammations were not a heterogeneous mixture of bacteria, but that they represented certain definite species with fixed morphological and biological characteristics; that the organisms, once having gained access to wounds, were followed by more or less constant alterations in the tissues and retardation of the processes of healing. From different conditions of suppuration he isolated four different species of bacteria, which he designated by the names staphylococcus pyogenes aureus, staphylococcus pyogenes albus, streptococcus pyogenes, and micrococcus tenuis. Most important in Rosenbach's work was the isolation of these organisms in pure cultures, and the establishment of their relation to acute suppurative processes, particularly the suppurative processes of bone—*i. e.*, infectious osteomyelitis. Through inoculations into lower animals he demonstrated that cold abscesses, from which the pyogenic cocci described by him were absent, were most commonly tuberculous in their nature.

Almost simultaneously with Rosenbach, Passet,[7] in 1885, confirmed throughout Rosenbach's work, and in addition described more minutely the pyogenic organisms, particularly their reactions to chemical and thermal influences, and their behavior under varying methods of cultivation. Passet's description of the specific pyogenic organisms was so complete that there has been but little of value added to it since. He showed that if these organisms, especially the staphylococcus pyogenes aureus, are injected directly into the circulation of rabbits, there always results the appearance of miliary abscesses throughout the body, and that the kidneys are the organs most frequently and to the largest degree affected. It was his belief that the three pyogenic cocci, the staphylococcus pyogenes aureus, albus, and citreus, were modifications of the same organism.

With the publication of Passet's work the question involving the etiology of suppurative processes seemed for the time to have been effectually settled, and for a period following, the pyogenic cocci of Ogston, Rosenbach, and Passet were believed by most observers to be the specific agents concerned in the production of these conditions. With the more general application of bacteriological methods, however, and the study of the manifold conditions coming under the eye of the pathologist, the physician, and the surgeon, results were now and then obtained that did not accord with the view commonly held in regard to the specific relation of the pyogenic cocci to all forms of suppuration. Evidence began to

accrue that pointed to the possibility of there being other organisms not grouped among the pyogenic bacteria which, under special conditions, could likewise produce in the tissues to which they had gained access conditions in all respects similar to those following the invasion of the specific pus-producing organisms.

So convincing is the evidence pointing to the relation of these organisms to the suppurative processes in which they are found that Verneuil[9] has suggested a classification of abscess-formations into those of a simple nature, those resulting from the presence of the ordinary pyogenic cocci in the tissues, and those of an "infectious nature," those occurring in the course of infectious diseases, and which in many instances are due to the activities of the specific causative element of the disease which is not normally pyogenic in its character.

The relation of these organisms, not normally pyogenic, cannot, however, be limited to abscess-production, for as has been repeatedly seen, they may be concerned in the production of suppurative processes generally. A striking illustration of the manifold inflammatory conditions that may result from the presence of these organisms in the tissues is afforded by some of the more recent contributions to the subject.

For example: The typhoid bacillus has been found by Welch[10] in pure culture in osteomyelitis of the ribs following typhoid fever; by Destrée,[11] in acute purulent otitis complicating typhoid fever; by Raymond,[12] in pure culture in abscesses of the soft parts in the course of the same disease; by Valentini,[13] in pure culture in the pus of empyema accompanying typhoid fever; and by A. Fraenkel,[14] in the fibro-purulent deposit of a localized peritonitis occurring four and a half months after the appearance of typhoid fever. Upon the relation of this organism to the suppurative processes in which it is found, Orloff,[15] as a result of experimental studies bearing upon this point, expresses the opinion that, where it is present in pure culture it must be looked upon as the causative agent in the production of the process, and that the pathological condition is not in these cases a result of mixed infection as some suppose.

In twenty cases of acute peritonitis examined by A. Fraenkel,[16] the bacterium coli communis was found to be the only organism present in nine of them; Welch[10] found the same organism in pure culture in fifteen different inflammatory conditions; Veillon and Jayle[17] found it in pure culture in the pus of a liver abscess accompanying dysentery; Gilbert and Girode[18] found it in pure culture in two cases of purulent inflammation of the gall-bladder and ducts; and Tavel[19] detected it in a hæmatoma following upon operation for the enucleation of a cyst of the thyroid.

The diplococcus pneumoniæ has likewise been found in pure culture in a variety of suppurative processes: in abscess of the soft parts, by Haegler;[20] in purulent infiltration of the tissues about a fracture of the ribs and of the hip, by Netter and Mariage;[21] by Steinhaus,[22] in purulent cerebro-spinal meningitis; by Picquet and Veillon,[23] in suppurative synovitis of the knee-joint; by A. Fraenkel,[16] in a case of acute peritonitis; by Zaufal,[24] in acute inflammation of the middle ear; and in acute fibrinous peritonitis, by Welch.[10]

Roswell Park[25] has detected the micrococcus tetragenous as the only organism present in an acute abscess; Kepper[26] has found the same organism in pure culture in an abscess of the soft parts about the lower jaw; Karlinski[27] has seen it in pure culture six times in inflammations of different characters, and Steinhaus[22] believes it to be one of the causes of suppuration in man.

In addition to these the bacterium lactis aërogenes has been found twice in pure cultures in acute peritonitis; Senator[28] describes a bacillus not yet identified as the only organism present in the pus of a suppurating tonsil; Pellizari[29] found only the gonococcus of Neisser in three cases of peri-urethral abscess; Karlinski[27] found the bacillus pyogenes fœtidus in three different inflammatory conditions, and Welch[10] detected in pure culture the bacillus pyogenes fœtidus in an abscess containing bad-smelling pus; the proteus Zenkeri in an ovarian abscess with purulent salpingitis; a bacillus apparently identical with the bacillus emphysematis maligni of Wicklein in an emphysematous cellulitis of the arm; a bacillus apparently identical with the bacillus enteriditis of Gärtner in the brain and other organs of an infant three months old with cerebral abscesses and meningitis following operation for imperforate anus; a bacillus not identified in a case of peritonitis, and a bacillus that could not be cultivated upon our ordinary media in one case of suppurative inflammation.

In consideration of such evidence as this it is plain that we can no longer adhere rigidly to the opinions formerly held regarding the etiology of suppuration, for we are forced to admit that while the pyogenic cocci of Ogston, Rosenbach, and Passet are the most frequent causes of this condition, still they are not the only causes, for, as we have seen, there exist other bacteria not normally pyogenic which, under conditions not entirely clear, may give rise to tissue-changes indistinguishable from those produced by the ordinary pus-organisms.

In seeking an explanation for this somewhat confusing and unexpected condition of affairs we naturally endeavor to discover the circumstances under which organisms of an entirely different character that are not

normally concerned in the production of suppuration may at times stand in direct causal relation to this process. As the earliest stage of spontaneous suppuration is characterized by a death of the tissues at and about the point occupied by the growing bacteria, it would seem reasonable to believe that there is a something produced by the bacteria in the course of their development that is directly detrimental to the tissues surrounding them; and we have abundant evidence in support of this opinion.

In endeavoring to study the relation between the necrotic conditions found and the substances believed to be concerned in their production, we find that these changes are not at all the specific results of bacterial poisons, but that they may be caused to occur from the presence in the tissues of substances that are in no way connected with bacteria or their life processes.

The well-known experiments of Councilman,[30] Uskoff,[31] Orthmann,[32] Dubler,[33] Grawitz,[34] Scheuerlen,[35] and many others since have demonstrated beyond doubt the possibility of producing suppurative processes, that are histologically and clinically identical with those of bacterial origin, by the subcutaneous introduction of irritants into the tissues; and careful examination of these abscesses shows them to be free from bacteria of any sort. Dubler (*loc. cit.*, page 97) remarks in regard to abscesses produced by the introduction of irritants into the tissues, that there are identically the same central area of necrosis and surrounding zone of inflammatory demarcation-tissue that are seen in abscesses of mycotic origin, and that throughout there is no marked difference between the two processes. If, as it appears to be, the conditions underlying the production of these changes are of a chemical nature, we then have a clue as to the means by which bacteria of an essentially different nature are concerned in the causation of the same process. Their products, though not of necessity similar, chemically possess, in common with irritants, a destructive action upon tissues with which they come in contact.

In recent times the chemical products of the growth of bacteria have been isolated and subjected to careful study, and it has been found that certain of them are poisons which, when separated from the bacteria by which they were formed, possess the power of causing tissue changes quite analogous to those following the growth of the bacteria themselves in the tissues, so that these poisonous albumins, albumoses, and toxalbumins, as they are called, have been held to be the means by which the invading organisms destroy the tissues and produce the pathological results.

Another standpoint from which the subject may be viewed is that brought out in the experiments of Buchner[36] and his pupils in Germany, which seem to demonstrate that the tissue changes characterizing these processes result less from the action of poisons produced by the growing bacteria than from the poisonous properties of the protoplasm of which the bacteria themselves are composed.

The question now arises: Why is it that one species of bacterium or one form of chemical compound does, while another does not, induce emigration of leukocytes with production of suppuration when introduced into the tissues of the living animal body?

In regard to the emigration of leukocytes toward a point in the tissues at which foreign matters are deposited, it has been shown by Leber,[37] Massart and Bordet,[38] and others, that in their relation to these substances the leukocytes follow the same general law of attraction and repulsion that Pfeffer has shown to govern the movements of motile bacteria when exposed to the influence of special chemical substances.

This singular and interesting phenomenon of chemotaxis, as it is called, is a peculiar relation that motile, living protoplasm is seen to bear toward certain substances of a chemical nature; some chemical compounds attracting, others repelling the protoplasmic body.

In 1881 Engelmann[39] demonstrated the effect of light upon the movements of motile bacteria. Not only the rate of motion, but likewise the character, and direction in which the movements were made, were influenced by it. These results he believed to be due to alterations in the chemical constitution of the medium in which the organisms were located, by the light to which it was exposed. He found also, under the influence of light, the effect of which was localized to definite parts of the fluid, that here there was, in particular cases, a production of oxygen as a result, and that the bacteria invariably moved themselves from the point in the fluid at which there was a scarcity of this gas to that where it was more abundant.

In 1887, Pfeffer[40] demonstrated the fact that motile bacteria, when subjected to the influence of chemical substances of different nature, were in some cases attracted toward it, and in other cases caused to retreat from it; in other words, the chemical body possessed a positive or a negative chemotactic affinity for these bacteria. Similar chemotactic affinities for foreign bodies are possessed by the leukocytes of the tissues in a high degree, and many substances, particularly those of bacterial origin, are claimed to possess constant positive or negative chemotactic affinities from these cells.

Leber,[37] Massart and Bordet,[38] and Gabritchewsky,[41] have shown that

an emigration of leukocytes, with subsequent aggregation into suppurating foci, follows constantly the introduction into the tissues of materials possessing positive chemotactic affinities.

It is claimed by Buchner that the products of bacterial growth, the ptomaïnes, toxines, and toxalbumins, are only nerve-poisons with but little or no power of inducing emigration of leukocytes, whereas the bacterio-protein, the plasma contents of the bacterial cell itself, possesses this property in a high degree. He found that subcutaneous injection of a few milligrammes of the proteid material extracted from the body of the bacillus pyocyaneus produced a condition possessing all the clinical and pathological characteristics of an erysipelatous inflammation. He has isolated this bacterio-protein from seven different species of bacteria, and finds that in all cases subcutaneous injections of these substances are followed by the appearance of suppurative inflammations of varying degrees of intensity.

From studies that have been made upon the behavior of bacteria in the tissues we know that the result is frequently an emigration of leukocytes toward them, and it seems fair to assume that this induction of wandering cells, with consequent formation of pus-points, is the result in some cases of the positive chemotactic affinities of poisons eliminated by the growing bacteria, while in others it occurs where dead bacteria are undergoing disintegration and the poisonous activities of the proteids, of which they themselves are composed, are in operation.

I doubt if we are justified in recognizing, as Buchner would have us do, these two causative factors as distinct the one from the other. We are not as yet in a position to say definitely to which of these influences the death of the tissue is due, or, indeed, that it is, when occurring spontaneously, the result of the action of the one to the exclusion of the other. For the present it would hardly be wise to make such a rigid distinction as suppuration occurring from poisons produced by living bacteria and that resulting from the poisonous proteids of the bacteria themselves. There is no reason for believing that all the individual bacteria that may have gained access in one way or another to the tissues retain their vitality and are actively concerned in the suppuration that results from their presence; on the contrary, evidence favors the belief that in the conflict normally seen to occur between invading organisms and healthy tissues a fair proportion of the invaders are rendered inactive. In some instances all, in others only a portion of the invading organisms may be killed. In the former case infection fails; in the latter, the infection that results may be either complete or partial—that is to say, there may be a true septicæmia with but little local reaction, or there may be an intense

local suppuration with but slight evidence of general infection. In the latter condition it is reasonable to assume that both the poisonous products of growth of the living bacteria and the poisonous activities of the proteid constituents of the dead and disintegrating bacteria are together concerned in producing the primary local necrosis of the tissues and the subsequent accumulation of leukocytes about it.

A further support for the opinion that we are not justified in considering these two factors as etiologically distinct in the production of these processes is brought out in the experiments of Rodet and Courmont,[42] who found that animals normally refractory to infection by the pyogenic organisms would become constantly susceptible if the normal resistance of their tissues was first diminished or destroyed by injecting into them the soluble products of the growth of these bacteria as obtained from artificial cultures.

Another point of considerable significance in explaining the process is an observation commonly made in the course of experiments upon the typical septicæmias of lower animals. Among the pathogenic bacteria are certain species which, when introduced into the tissues of susceptible animals under normal conditions, result in the production of acute general infection with but slight visible reaction at or about the point of inoculation. Anthrax and the septicæmia resulting from inoculations with the diplococcus pneumoniæ are conspicuous examples. In the course of this work, however, it not uncommonly occurs that there will arise conditions, with some of which we are acquainted, others not known to us, that influence either the resistance of the animal tissues and cause it to become increased in power or diminish the virulence of the organisms through degenerative changes in the protoplasm of which they are composed. Under these circumstances the results of inoculation are likely to be modified, and, instead of the usual relation between the number of bacteria in the blood of the animal—that is, the intensity of the general infection—and the local reaction at the seat of inoculation, there is a marked decrease in the general expression and an increase of the local condition, so that in these cases there may be at and about the seat of inoculation degrees of reaction extending from simple œdema to an intense, widespread, fibro-purulent infiltration.

After inoculation of susceptible animals with the bacillus of anthrax, which normally produces a typical septicæmia, particularly in rabbits, there occurs at the seat of operation but little reaction other than an œdematous condition of the surrounding tissues; but if these organisms be subjected to influences that bring about degenerative changes in the protoplasm of which they are composed, with consequent attenuation of

their normal virulence, their introduction into the tissues is commonly followed by local lesions that may be accompanied by suppuration, and but rarely or not at all by general infection.

Whether this local condition that is characterized by an emigration of leukocytes to the part at which bacteria are located indicates an effort on the part of the tissues to combat and prevent the inroads of disease-producing elements or not, it is impossible to say, but the fact remains that whenever it does occur, as a result of inoculation of organisms that under favorable conditions produce intense general infection, the general constitutional expressions are of much less intensity than where no such local reaction is present. Certainly there are causes at work in these conditions that attract the wandering cells of the body to a definite location, and this spot is the point at which foreign poisonous substances have been deposited. The variations in the intensity of these local processes that are seen to vary with the character of the agents employed, speak strongly for a direct connection between the effect found and what we believe to be the cause.

Welch[10] has called attention to that point, and cites in illustration the results of inoculation with the two varieties of the swine-plague bacillus, varieties that appear to represent modifications in the pathogenic powers of the same organism. Inoculations with one of these organisms result in death of the animal in from twelve to eighteen hours, with only slight local reaction and many organisms in the blood, while the other causes an extensive purulent infiltration at the point of inoculation, with but few organisms in the blood. These differences, he believes, uphold the idea that the local condition expresses an effort on the part of the tissues toward resisting infection, and that the more intense and extensive the local reaction the fewer organisms, as a rule, are to be found in the blood—that is to say, the less pronounced will be the process of general infection. In support of the same opinion it has been shown experimentally that if animals are inoculated with the organisms concerned in the production of certain forms of acute septicæmia, and immediately following again inoculated at the same point with one of the organisms capable of producing a localized infiltration of leukocytes, no general septic condition will result. It is likewise a common experience, in experiments with certain of the organisms concerned in producing the so-called "hemorrhagic septicæmias," that there does occur after inoculations with the same culture of the same organism in some cases typical general septicæmia with but little change at the seat of inoculation, in others an intense local condition with but limited general expression.

This relation between local and constitutional reaction is frequently to

be seen after subcutaneous inoculation of pigeons with the organism of chicken cholera. Normally the result is general septicæmia with trifling local manifestations. But now and then the degree of intensity of the general septicæmia may be extremely limited, while the point of inoculation may be marked by an extensive purulent infiltration of the tissues round about it.

If, as it seems reasonable to suppose, the local aggregation of leukocytes expresses an effort on the part of the tissue to prevent infection, we are justified in expecting, as a result of the combat, the destruction of a certain number of the invaders, and closer inspection of the local condition points to the accuracy of this belief.

Microscopic examination of the tissues at and about the point of inoculation, particularly where the local reaction is intense, always results in the detection of bacteria advanced in the stage of involution and degeneration, conditions resulting most probably from the detrimenta influences of either the cells or fluids of the tissues in their effort to resist infection. This germicidal activity of the fluids of the body has been too much discussed in recent times to require rehearsal here; it suffices to say that it does occur, and not only may be seen in the body of the living animal, but can be demonstrated in the serum from the blood of the animal outside of the body. This power, however, is apparently limited in degree and does not in every case result in complete destruction of all the invaders, unless the animal possesses absolute immunity against the pathogenic activities of the organisms employed. The conditions that have been cited appear to indicate a partially effectual effort on the part of the tissues to establish this refractory state.

Certainly there must be an explanation for the fact that organisms normally concerned in the production of acute, general, septic conditions may at one time run their normal course when introduced into the body of a susceptible animal, while at another their pathogenic activities find their most conspicuous expression in tissue changes of a local suppurative character. These discrepancies seem to be best explained upon two grounds already mentioned—one of which assumes variations in the power of the tissues to resist invading organisms, while the other depends upon fluctuations in virulence and alterations in the ability of the invading organisms to overcome the tissues.

Where the organisms possess their full virulence the tissues of susceptible animals succumb and the general constitutional expression of the process results, but where from unfavorable surroundings the virulence of the organism is weakened, or, under conditions not known to us, the destructive activities of the tissues are increased, then a disinte-

gration of the organisms at the point of inoculation is frequently seen. With this disintegration there occurs, as is now believed by some, an attraction of leukocytes toward the proteid substances set free from the bodies of the bacteria themselves, and a consequent establishment of a process of suppuration.

Time will not permit of my reviewing in detail the many contributions that have been made to this important subject. It suffices to say that studies, especially of Buchner[36] and his pupils in Germany, and of Prudden and Hodenpyl[43] in this country, have proved that the substances comprising the bodies of certain bacterial cells—the bacterio-proteins—when introduced into the tissues, possess the property of attracting leukocytes toward them, a phenomenon so analogous to that observed by Pfeffer when motile bacteria were exposed to various chemical substances as in all probability to be identical with it; and, though this phenomenon of chemotaxis is as yet somewhat in obscurity, still it seems to be the source to which we must look for an explanation of certain of the undecided points in pathology, particularly the manifold conditions that underlie the production of suppuration.

From the evidence that has been presented to you in this paper it is plain that suppuration cannot be considered a specific process in the same way that tuberculosis, diphtheria, and anthrax are specific processes, but that the causes underlying it are manifold: in most cases being the result of the presence in the tissues of the common pyogenic cocci; frequently following the invasion of the tissues by organisms not normally pyogenic in character; produced experimentally by a variety of irritating substances without the presence of bacteria, and by the poisonous products of the growth of bacteria, and, finally, following the introduction into the tissues of the proteid substances that make up the body of the bacteria themselves.

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April 28, 1892.

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9. *Annual Address of the President to the Pathological Society.*

By Dr. Arthur V. Meigs.

A by-law of our Society directs that the President shall "at the annual meeting for elections in October of each year make an address to the Society, in which he shall describe (sum up) the work of the Society for the past year, and make such suggestions in regard to its labors and the mode of conducting them as may seem to him desirable."

In accordance with this mandate I shall briefly review the work of our Society during the past year, and call your attention to what might

be done to make our labors more valuable, both to ourselves and to others.

During the year there have been eighteen stated meetings held, this being the full number directed by the by-law, no meeting having failed for the lack of a quorum. There have been shown about seventy-one specimens of disease, some of them being of great interest. No conversational meeting with a specially prepared address was held in October, owing to a failure to make preparation for it. At the April conversational meeting Dr. A. C. Abbott delivered an address upon "The Factors Concerned in the Process of Suppuration." The subject is one of great importance to the student of pathology, and the address was scholarly, embodying the most recent views, and containing much that was unknown to many of us. The meeting was well attended and much interest manifested. In addition to this, which is the regular rule of the Society, the greater portion of two meetings was devoted to the discussion of subjects which had been previously specially selected. Upon one occasion the discussion was of "Tubercular Lesions of the Bones and Joints," and it was introduced by Drs. John Ashhurst and De Forest Willard; in the other it was "Lesions Secondary to Pulmonary Tuberculosis," and Drs. John Guitéras and J. H. Musser delivered addresses. On both evenings the attendance was full and the interest elicited was most gratifying.

Having thus sketched in outline the work of the Society during the past year, it will not be out of place perhaps for me, as your presiding officer, to indicate what has struck me as possible to be done in the way of improvement. Our proceedings at the stated meetings are too formal and too informal. Specimens presented, if such presentation is to accomplish the greatest amount of good, should be accompanied with more carefully prepared reports of their appearances and peculiarities, and microscopical examinations should be more frequently made. On the other hand, it has struck me that it would be much to the advantage of the Society, and would increase the amount of knowledge we would acquire by attending its meetings, if verbal reports were made of specimens or subjects of interest or obscurity coming under the notice of members. Such communications, however, should either not be reported at all, or merely sketched in outline by the recorder. For my own part, I see nothing in the by-laws which need be altered to enable us to do this, and I think members of the Society might often be tempted to open subjects in this more informal way which they would feel were too difficult or too little understood by them to be introduced by formal papers. Such discussions should, of course, be restrained within the

bounds of debate by your presiding officer, or our proceedings might degenerate into mere informal conversation.

A year's study and observation of the working of our Pathological Society as its President has brought me to a still greater realization of the important position held by one special branch as a necessary integral part of the science of medicine. It has often struck me as astonishing how great is the interest taken by practical physicians in pathology. Some of its problems are so abstruse and its study requires, as years go by, so great a degree of specialization, that it would not at first thought seem unnatural that the every-day physician should lose interest in it. Many of the profession, both among its older and younger members, take an interest in pathological research and results which are much more than commensurate with any possible return they could get from the time bestowed upon them. The direct pecuniary rewards in most instances are nothing, or even less than nothing, for many of us must have expensive books and instruments and never receive any perceptible return. The original source of the love for pathological study amongst us is that secured to anatomy—and to anatomy alone does it yield the precedence—upon the science of pathology is based the whole superstructure of modern medicine as it exists to-day.

I must not close without expressing to the members of this Society the deep sense of my appreciation of the compliment which was paid me and the confidence shown in me when I was elected to preside over you during the past year. *October* 13, 1892.

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10. *The giant cell of tubercle.*

Read by Dr. Joseph McFarland.

The first change which the presence of a tubercle bacillus produces in a tissue[1] is multiplication of its fixed connective-tissue cells, which increase in size, divide by karyokinesis of the nucleus, and thus give origin to the so-called epithelioid cells, which may be looked upon as the essential elements of the tubercle, always being present at some stage of its development. The epithelioid cells are large, have an irregularly spindle shape, possess a considerable amount of protoplasm, and contain one, two, and occasionally several oval vesicular nuclei. The nuclei are finely reticulated, contain numerous fine granules, and usually a nucleolus.

[1] Baumgarten: Pathologische Mykologie, 1890, Bd. ii.

Fig. 1 Giant Cells
1. Peripheral or circumferential.
2. Polar.
3. Equatorial.

Fig. 2 Hyaloid Degeneration
a. Giant cell from a miliary tubercle of the liver.
b. Giant cell from a cheesy lymph gland.

Fig. 4. Amoeboid or corpuscle of the giant cell described by Brodowski.

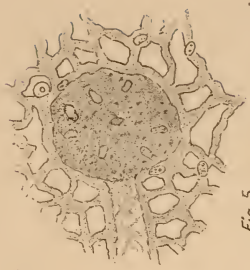

Fig. 5. Origin of giant cell as described by Schuppel.

Fig. 3. Evolution of a giant cell from a bile duct. From Arnold.

At the same time that the epithelioid cells are accumulating, a second cellular factor usually appears in the form of a rather widespread (depending upon the character of the tissue) infiltration of *small round cells*, which are very probably leukocytes, and may be few in number or very numerous. They usually form a zone surrounding the epithelioid cells, but are not circumscribed by any distinct line, and sometimes distribute themselves so evenly through the tubercle, among the epithelioid cells, and so preponderate over them in number as to make the tubercle appear to consist solely of them. When caseation begins, the small round cells are the first to metamorphose, and, as they change into a granular detritus, the epithelioid cells again come into prominence for a time. Little by little these also show signs of degeneration ; the nucleus stains but faintly, then not at all, and finally the entire cell loses its configuration, becomes yellow, granular, and disappears in the cheesy mass. In its formation and growth by cellular proliferation, as described, the tubercle occupies considerable space at the expense of the normal cellular elements of the tissue, which atrophy and disappear, leaving the more resistant fibrous stroma to form the framework of the tubercle. Sometimes when caseation is advanced this fibrous stroma remains prominent, and gives the tubercle a reticulated appearance. Tubercles are constantly avascular, occlusion of neighboring capillaries and small vessels occurring by proliferation of their endothelium and by thrombosis.

The tubercle bacilli have a very constant relation to the tubercle, and usually are most numerous in the healthy, actively-growing periphery, while few are present in the degenerating centre. They are not only found occupying the lymph spaces between the cells, but also are found *in* certain of the cells. The small round cells are usually without enclosed bacilli, while the epithelioid cells contain large numbers, hence, the tubercle bacillus being possessed of no individual movement, the latter cells are supposed to have amœboid movement, and are classed with the "phagocytes." Bacilli which occupy the degenerated centre are rarely numerous, and usually exhibit the peculiar beaded appearance which is supposed to indicate their involution.

In the majority of tubercles a third cellular element presents itself, viz., the GIANT CELL, which for so long a time was considered characteristic of tubercle. The giant cells are large, irregular, protoplasmic bodies measuring from 14.6 μ to 138.8 μ, and containing from half a dozen to several hundred oval, slightly flattened, vesicular nuclei of equal size. In the smaller cells the nuclei show no tendency to a definite arrangement, but in the larger cells they are usually characterized by a circumferential, polar, or equatorial distribution.

The giant cells often appear to lie in distinct open spaces, which are probably dilated lymph-spaces, and sometimes are surrounded by an envelope of fibrous tissues—the mantle of Langhans. As the cheesy degeneration of the tubercle progresses, the giant cells, which usually lie at its periphery, also show signs of beginning degeneration. The protoplasm of the anuclear areas becomes slightly yellowish, finely granular, and somewhat opaque. The nuclei appear to shrink and to assume a club shape; finally they refuse to stain, become indistinct, and are lost in the granular *débris*.

They also seem to suffer a second form of metamorphosis, which may be described as *hyaloid*, and which is more common in giant-cell sarcoma than in tubercle.

In this form of degeneration colorless droplets, resembling vacuoles, appear in the protoplasm, increase in size and number until the entire cell is destroyed, and only a collection of drops remains in the space once occupied by the cell.

The protoplasm of giant cells frequently contains foreign particles—red blood-corpuscles, pigment granules, tubercle bacilli, dust, etc.—and hence are phagocytic, and to some extent amœboid. They almost always contain tubercle bacilli, and Weigert,[1] who gave this fact special attention, found that one hundred giant cells contained one hundred and fifty-three bacilli. In seventy he found a single bacillus to the cell; in eighty-eight the bacilli occupied the nuclear zone; in six, bacilli were found in the anuclear centre. Several cells were found to contain so many bacilli that it was impossible to count them.

From this and other studies of the subject, we find the distribution of bacilli in the giant cell to be approximately the same as in the tubercle itself—*i. e.*, they lie in the properly-nourished periphery rather than in the degenerated centre. Occasionally they are found in the cheesy centres of the giant cells, but in these cases degeneration is apt to affect the bacilli themselves.

The evolution of our knowledge of the giant cell is an interesting study, and, in following the literature of the subject, one can but remark upon the different theories which have been advanced to explain its origin, and the efforts which have been made to attribute to it a definite function.

Rokitansky,[2] in 1855, makes the first mention of the giant cell in tuberculosis which I have been able to find. Believing in endogenous cell formation, he thought the cells to be "Brutzellen" or "Mutterzel-

[1] Deutsche medicinische Wochenschrift, August 27, 1885, p. 599.
[2] Lehrbuch der pathologische Anatomie, Wien, 1855, Bd. i. Ss. 294, 295.

len," and, mistaking the nuclei for young cells, believed that the small cells of the tubercle originated in the giant cells.

Virchow[1] spoke of finding round, yellowish, opaque accumulations of finely granular appearance, not rarely surrounded by a thick and dense connective-tissue layer, suggesting that transverse "sections of lymph vessels with thickened contents had been made." The peripheral arrangement of the nuclei suggested to him that the explanation of the occurrence of these unusual bodies might be found in proliferation of the epithelium lining lymph channels, but he was unable to satisfy himself of this.

Again,[2] he suggests their origin from connective-tissue corpuscles, but admits the possibility of their formation by other cells, as endothelium, epithelium, nerves, muscles, etc.

Langhans[3] called attention to the frequent circumferential arrangement of the nuclei. He did not agree with Virchow that they represented altered lymph vessels, because he found them scattered irregularly through peritoneal tubercles, which he studied, and never arranged in a continuous series, as should be the case if they actually were lymph vessels. In tubercular disease of the choroid he found that the giant cells contained pigment, and hence concluded their existence to be dependent upon the fusion of neighboring stellate pigmented cells, or the overgrowth of individual epithelioid cells.

Klebs[4] continued to incline toward the view of their lymph-vascular origin, and suggested the probability of their formation by the coagulation of albuminous bodies in the vessels, especially when small round cells were incorporated with them.

Köster[5] thought they arose from the endothelium of vessels.

Schüppel[6] designated them as *protoblasts*, or cellulo-genetic bodies, formed by the coagulation of fibrinous masses in the bloodvessels.

His idea seems to be that these fibrinous masses become cells by the spontaneous generation of nuclei. From the protoblasts new cells were given off into the surrounding tissue. As the cells had very irregular outlines, he thought them amœboid.

Brodowski[7] said: "With a word my observations lead me to the

[1] Die krankhafte Geschwülste, 1863, Bd. ii. SS. 640, 641.

[2] Virchow's Archiv, Bd. xiv.

[3] "Ueber Riesenzellen mit wandständigen Kernen," Virchow's Archiv, 1868, Bd. xlii.

[4] "Beiträge zur Geschichte der Tuberculose," Virchow's Archiv, Bd. xliv.

[5] "Ueber fungöse Gelenkentzündung," Virchow's Archiv, Bd. xlviii.

[6] "Ueber die Entstehung von Riesenzellen im Tuberkel," Archiv der Heilkunde, 1872, Bd. xiii.

[7] "Ueber die Ursprung sog. Riesenzellen und über Tuberkel im allgemein," Virchow's Archiv, 1875, Bd. lxiii.

conviction that the formation of the so-called giant cells is brought about by the abnormal activity of the bloodvessels. This abnormal activity characterizes itself, on the one hand, by hypertrophy of certain parts of the protoplasmic germs of these vessels, and through the formation of an unusually large number of nuclei in the hypertrophied parts of the protoplasmic germ; . . . on the other hand, this deviation is characterized by cessation of the development of the misformed germ."

" . . . In spite of this still-stand, one can observe in the middle of the capillary vessels protoplasmic bridges connecting with the giant cells, and also in the cells themselves—one might say fragmentarily—this or that appearance which suggests the manifestation of the development of bloodvessels." He called this appearance "granuloma gigantoangioblastium."

Tizzoni[1] thought giant cells to be altered lymph vessels.

Jacobson[2] studied the granulations of normally healing wounds, and emphasizes the fact, pointed out by Billroth as early as 1856, that giant cells form a pretty constant entity in granulations.

Weiss,[3] like Jacobson, diverted from the tubercle itself and studied giant cells in their general relation to disease. Experimentally introducing into the cellular tissue fibres of cotton, etc., his results and conclusions are as follows:

1. The giant cells (which he always found surrounding the foreign bodies) are formed by the confluence of the protoplasm of neighboring cells.

2. The cells which thus originate the giant cells are granulation cells.

3. The giant cells do not change either into connective tissue or bloodvessels, but always undergo fatty metamorphosis, even when the conditions of life are best.

He thought it impossible that the giant cells originated from mononuclear cells by multiplication of the nuclei, because he found the foreign bodies *in* the giant cells. This proved to him that the cells were formed around them, and, as in many places he found groups of leukocytes surrounding the foreign body, his inference was that the giant cells were formed by the fusion of these.

[1] "Ein Beitrag zur Lehre von der Hodentuberculose," Virchow's Archiv, 1875, Bd. lxiii.

[2] "Ueber das Vorkommen von Riesenzellen in gut granulirenden Wunden," Virchow's Archiv, 1875, Bd. lxv.

[3] "Ueber die Bildung und die Bedeutung der Riesenzellen, und über epithelartige Zellen welche um Fremdkörper herum in Organism sich bilden," Virchow's Archiv, 1876, Bd. lxviii.

Lübimow[1] thought that giant cells were formed by the addition of protoplasm to, and the multiplication of, the nuclei in epithelioid cells, and thought that they occupied the lymph spaces.

Weigert[2] also believed that giant cells originated from the epithelioid cells by multiplication of the nuclei, and gave us the first satisfactory explanation of their eccentric nuclei by calling attention to the fact that the cheesy change which occurs in the protoplasm of the cell is a variety of coagulation necrosis, a local death of the cell, and that the nuclei, which possess the power of moving within the cell, seek the still normally nourished, healthy protoplasm of the periphery.

Arnold[3] thought that in miliary tuberculosis of the liver and kidney giant cells were formed by the confluence of neighboring epithelial cells lining detached portions of bile ducts and uriniferous tubules.

In lymph glands he viewed the giant cells as originating either from the confluence of the endothelial cells of the vessels or from the confluence of neighboring epithelioid cells. Red blood-corpuscles occasionally met were held by him to have been closed in at the moment of fusion. He also thought it possible for giant cells to increase in number by division.

Hamilton,[4] in studying the use of de-mineralized sponges introduced into wounds for the purpose of supplying a framework upon which new vessels, etc., might be formed, found many giant cells in the meshes of the sponge. At first he thought these were spongioclasts, or cells like osteoclasts, produced by the organism for the purpose of removing the foreign structure, but later was obliged to conclude that they were produced purely by the irritative presence of the foreign body.

Waldstein[5] regards the giant cell as the product of the confluence of neighboring cells which seem to undergo a peculiar liquefaction. He offered no explanation of this phenomenon or for the peripherally-arranged nuclei which he mentions.

Marchand[6] found that giant cells could be produced by the subcutaneous introduction of minute particles of silk, etc., and that the foreign particle was usually enclosed in the giant cell. If the cell was small,

[1] "Zur Frage über die Histogenese der Riesenzellen in der Tuberculose," Virchow's Archiv, 1879, Bd. lxxv.

[2] "Ueber pathologische Gerinnungsvorgänge," Virchow's Archiv, 1880, Bd. lxxxii.

[3] "Beiträge zur Anatomie des Miliartuberkels." Virchow's Archiv, Bd. lxxxii., lxxxiii., and lxxxv.

[4] "On Sponge-grafting," Edinburgh Medical Journal, 1881, vol. xxvii., part i.

[5] "Zur Kentniss der Tuberculösen Erkrankungen des Hodens," Virchow's Archiv, 1881, Bd. lxxxv.

[6] "Ueber die Bildungsweisse der Riesenzellen um Fremdkörper, etc.," Virchow's Archiv, 1883, Bd. xciii.

the body was apt to occupy one pole, but if large, the body was more likely to be in the centre. He concluded that the giant cells were produced by the growth of the epithelioid cells, which in turn arose from the endothelium of the vessels and lymph vessels. When iodoform was introduced with the bits of foreign material, no giant cells were observed, hence he concludes that iodoform has a peculiar inhibitory influence upon their growth. His investigations showed that round cells gathered around foreign matter almost immediately. In twenty-four hours epithelioid cells were found, in ten days small giant cells could be noted, while about fifteen days were required before large giant cells with circumferential nuclei were numerous. These experiments were all done with aseptic precautions.

Weigert,[1] in 1885, repeats his former belief that the giant cell represents a partial caseation of a growing cell. The central necrotic portion being unfitted for the sustenance of either nuclei or bacilli, they had to move toward the more healthy peripheral part of the cell where the nutrition is good. He thought it possible that the irritation caused by the presence of the tubercle bacillus was sufficient to cause the epithelioid cells (which generally contained bacilli) to increase in size, to multiply their nuclei and thus generate large cells, which did not divide into smallar cells because retarded by beginning degeneration at some point. He considered the possibility of giant cells being formed by the fusion of neighboring cells, and also mentioned Arnold's view of their epithelial origin as possible, but highly improbable, and only occurring where simultaneous coagulation necrosis occurred in neighboring cells. As often as an eccentric arrangement of the nuclei of giant cells occurs, whether in the cells in tubercle, granulations, foreign-body inoculations, etc., he considers it dependent upon the partial death of the protoplasm of the cell.

Naegeli[2] introduced the clotted blood of a pigeon beneath the skin of other pigeons, and found the production of giant cells around the foreign body to be dependent upon the presence of micro-organisms. In seventeen experiments, which he conducted without precautions regarding antisepsis or even cleanliness, he found giant cells present in all but five, and was able to demonstrate the presence of micro-organisms. In twenty carefully aseptic experiments no micro-organisms and no giant cells were found. When present, the giant cells were always external to the clot—

[1] "Zur Theorie der tuberculösen Riesenzellen," Deutsche medicinische Wochenschrift, 1885, p. 599.

[2] "Ueber den Einfluss der Pilze auf die Bildung von Riesenzellen mit wandständigen Kernen," Archiv für experimentelle Pathologie und Pharmakologie, 1885, Bd. xix.

i. e., between the clot and the skin. He thought it improbable that all of Marchand's carefully-conducted experiments were septic, yet differs from him altogether in his results.

Metschnikoff[1] introduced a new era when he began to apply a protective purpose to the phenomena of phagocytosis. His studies upon the amœba, daphnia, frog, and then upon the cells of the human body, led him to conclude that certain of the bodily cells were capable of digesting micro-organisms and thus rendering them incapable of further mischief in the body. He attributed this same function to the giant cells of tubercle which he sees ingest and digest the tubercle bacilli. He even thought it probable that the giant cell *secreted an acid which operated destructively upon the bacilli.*

Holmfeld,[2] Buchner,[3] Fudor,[4] and Nuttall,[5] to whose researches, however, but little reference can be made, must be quoted as introducing a new line of thought to combat Metschnikoff. According to these and other observers, the blood, plasma, and serum possess the power of destroying bacteria, and phagocytes, in their eyes, become but the "charnel-houses" in which the dead repose.

Metschnikoff[6] investigated the giant cells of tubercle with special reference to their phagocytic tendency, and using an animal known to be scarcely at all susceptible to tuberculosis—a variety of the marmot known as the ziesel—injected into the abdominal cavity a virulent culture of tubercle bacilli.

The animal did not waste and die as susceptible animals do, but, on the contrary, fattened and lived on in a fair way toward recovery. When killed, special attention was given to the liver and spleen, where no tubercles were found, but where a profuse development of epithelioid cells, with all the intermediate steps between them and the large number of giant cells present, was easily observed. The bacilli were found enclosed in the protoplasm of the epithelioid and giant cells. None were observed in the epithelial cells or in leukocytes. He found, by staining the bacilli with fuchsin, that some stained very well, giving

[1] "Ueber die Beziehung der Phagocyten zu den Milzbrandbacillen," Virchow's Archiv, Bd. xcvii.

[2] "Ueber Immunität und Phagocytose," Fortschritte der Medicin, 1887, No. 13.

[3] "Ueber die Bakterientödende Wirkung des freien Blutserums," Centralbl. f. Bakt., 1889, v. and vi.

[4] "Ueber die Fähigkeit des Blutes, Bakterien zu vernichten," Deutsche med. Wochenschr., 1887.

[5] "Experimente über die bakterienfeindlichen Einflusse des Thierischenkörpers," Zeitschrift f. Hyg., iv.

[6] "Ueber die Phagocytäre Rolle der Tuberkelriesenzellen," Virchow's Archiv, 1888, Bd. cxiii.

sharp, clear outlines; others stained poorly, and still others possessed a remarkable kind of surrounding capsule somewhat resembling the capsule of the pneumococcus, but of a yellowish color. Although he had studied a large variety of forms of the bacillus which were observed in old and degenerating cultures, yet he had never met this picture, to which he gave the name "yellow degeneration." Study led him to conclude that this was the bacillus as it appeared when caught in process of digestion by the giant cell, and he soon regarded the homogeneous yellow capsule as the swollen cell-wall of the bacillus acted upon by the substances secreted by the cell. Soon he concluded that the yellow granules which occupied some of the cells were not the expression of degeneration, but were instead the dead bodies of destroyed bacilli or droplets of the poison elaborated by the cell for their destruction.

The giant cells were irregular in shape, and occasionally presented processes ramifying among neighboring cells, which he noted and thought sufficient proof of their *actively* amœboid nature. He also observed some which were constricted at the centre, presenting an hour-glass shape, and concluded that the giant cells multiplied by division.

His theory of tubercle would therefore be that the bacilli cause multiplication of the connective-tissue cells of the part, producing epithelioid cells, which at once proceed to swallow the bacilli and destroy them. Some of the cells, by growth of the protoplasm and karyokinesis of the nucleus, become giant cells and the arch enemies of the bacilli. He is particular to tell us that the production of these giant cells is in no way irritative and dependent upon the presence of contained bacilli, for he has found giant cells which contained no bacilli. No, it is a purposeful activity of the cells having for its object the destruction of the bacilli. Seeing that in man, the guinea-pig, and other susceptible animals the giant cells seem prone to die and degenerate, he explains that the giant cells in these animals are too weak to combat the bacilli and are themselves overcome.

Weigert,[1] in answering Metschnikoff's theory, explains that lack of color is no proof of the degenerated condition of the bacilli, and points out that Baumgarten, who could not find any yellow degenerated bacilli, may have used a better method of staining than Metschnikoff, when all the bacilli would be stained an intense red, or he may have used an inferior method of staining, when degenerated bacilli might not have been stained at all. Again, he says staining bears no relation to viru-

[1] "Ueber Metschnikoff's Theorie der Tuberkelriesenzellen," Fortschr. der Medicin, No. 21, 1888.

lence, for, although most of our specimens are immersed in absolute alcohol, and so are surely dead, yet they stain exceedingly well. However, as anthrax bacilli stain best when most virulent, he is willing to admit that the same may be true of tubercle bacilli.

The hyaline cylinders by which the yellow degenerated bacilli are surrounded would, he thinks, more likely be degenerated portions of the cell deleteriously affected by the bacillary products than *vice versa.* Hyaline change is common in the cells of tubercle. Metschnikoff's assertion that giant cells are amœboid, because they have processes, he thinks ungrounded, although he says they likely are, as they are phagocytic. An hour-glass shape is no more a sign of division than a club shape or a triangular shape would be.

Instead of regarding the yellow granules of the cells as cellular secretions, he returns to his former view of coagulation necrosis, and regards it as the expression of local death of the cell.

Stschastny,[1] working independently of Metschnikoff, and sending the results of his own work to press before his article appeared, concludes from his experiments that the giant cells of tubercle are capable of exerting a destructive influence upon the bacilli. He objects to the view of Weigert and Baumgarten, that the granular centre of the giant cell is necrotic, and states that in the ziesel he never found it so. In many cells he found fragments of red blood-corpuscles and dark-brown pigment. He could trace completely the development of the giant cell from the epithelioid cell. In sparrows, which show the most acute form of tuberculosis, he never found giant cells, although the bacilli were distributed about in the tissues, and the epithelioid and granulation cells were full of them. His conclusions are:

1. The "wanderzellen" of the blood are phagocytes and eat up active bacilli.

2. Tubercle bacilli thus taken up may be carried to distant parts of the body and there set up tuberculosis.

3. Some of the leukocytes, by nuclear division or fragmentation, may change to giant cells.

4. Fixed connective-tissue cells which take up bacilli may become giant cells, but he did not see it in the ziesel.

5. Whether cells free of bacilli can change to giant cells is an open question.

6. The giant cells of susceptible animals suffer degeneration, those of immune animals do not.

[1] "Ueber die Beziehung der Tuberkelbacillen zu den Zellen," Virchow's Archiv, 1889, Bd. cxv.

7. The giant cells hasten the natural involution of the bacilli and produce dead and degenerated forms, which do not occur in cultures.

8. The primary epithelioid tubercle cannot be differentiated from the primary lymphoid tubercle. Both may arise from bacilli-containing leukocytes.

9. The theory of Metschnikoff and Roser is to be considered true and free from doubts.

Baumgarten's[1] beautiful studies of tubercle of the iris have done much to perfect our knowledge of the subject. He finds the primitive tubercle to consist exclusively of newly-formed epithelioid cells, produced by karyokinesis of the fixed connective-tissue cells. It may be asserted with positiveness that giant cells do not arise by fusion of neighboring cells, but do arise from epithelioid cells by nuclear division. He has seen mitotic figures in the nuclei of a giant cell. He deems Weigert's view of partial cell death a satisfactory explanation of the peripheral arrangement of the nuclei.

Prudden[2] has recently made some experiments of unusual interest by introducing *dead* tubercle bacilli into the living body. He finds at the point of lodgment a tubercle-like nodule develops, whose histology is almost exactly that of the disease when naturally acquired, except that no degeneration occurs. The dead bacilli are always found in the tubercle, and stain well. At first it would seem that the bacilli must be living, but it is impossible, as the cultures were boiled for long periods, and when the sterilized cultures were introduced into control rabbits and guinea-pigs, they did not develop tuberculosis. In these cases the giant cells and bacilli seem to bear no distinct relation to each other. Giant cells were always produced as in ordinary tubercles, but did not degenerate. The whole tubercle showed a tendency to heal, and as time passed the number of bacilli grew less and less.

After such a *résumé* of our knowledge of the subject it need scarcely be said that the giant cells are the result of an excessive growth of the epithelioid cells. Probably the same factors are concerned in their production as operate in producing the epithelioid cells, and, although Metschnikoff does not believe that the irritation of the cell by the tubercle bacillus is operative in the development of giant cells, the fact that a foreign body, be it a live or dead bacillus, a thread of cotton, a fibre of silk, a piece of glass or dust introduced into the tissue, is potent

[1] Loc cit.

[2] "Studies on the Action of Dead Bacilli in the Living Body," New York Medical Journal, June 6 and 20, 1891. "A Study of Experimental Pneumonitis in the Rabbit," New York Medical Journal, December 5, 1891.

in their genesis, would make it seem reasonable to suppose that irritation is the cause of this overgrowth.

The theory of Weigert, that the nuclei of the cell being movable within the cell seek that part of it best fitted for their nourishment, would account for the peculiar arrangement of the nuclei so often met. If the centre of the cell degenerated, the nuclei would be circumferential. If the cell were long drawn out in the longitudinal axis, and considerable of the centre died, the nuclei would seek the poles. If the poles metamorphosed, the nuclei would seek the median zone. The conditions of life which apply to the nuclei also apply to the bacilli, which are most numerous where the nutrition is best, and so are found at the periphery of the tubercle and at the periphery of the giant cell.

More difficult than the solution of the problem of the origin of the giant cell is that of its possible function. Does the giant cell of tubercle have a special function to perform?

We have seen that the giant cells often have enclosed in their protoplasm a variety of foreign bodies, such as bacilli, dust, pigment, red blood-corpuscles, etc. Metschnikoff, observing this, has suggested that they are produced for the purpose of devouring these articles and digesting as many of them as possible. There can be no question that the giant cell devours. Does it devour to destroy? We have seen how much of the success of his theory depends upon the bacilli noticed to suffer from the yellow degeneration described by Metschnikoff, and have also seen how questionable a test which depends upon staining must be. If his peculiarly colored bacilli are the result of improper staining, there probably is no such thing as yellow degeneration, and we remember that Baumgarten could not find it. If there is no yellow degeneration, there is no evidence that the giant cell operates upon the bacilli in any way differing from its action toward other particles. When we place these facts side by side with the discoveries of Buchner, Fudor, Nuttall, and others who have proved how destructively the blood itself acts upon micro-organisms, we find that the consumed bacilli may be treated in no way differently from silk and cotton filaments or other dead particles which have been devoured by the cells. We find, too, from the researches of Prudden, that in the tubercles of dead-bacilli origin giant cells are present.

Putting these things together, we find that it is not only questionable, but highly improbable, that the giant cell is a specially detailed policeman, whose duty it is to arrest, incarcerate, and execute the destroyers of health and cellular order.

The view which we must take of the giant cell of tubercle, like that of

giant-cells in general, is that it is a *cellular monstrosity, produced by an abnormal, irritative, reproductive effort of the epithelioid cell;* that it is a monster in size and shape, but still partakes of the amœboid and phagocytic nature of the parent mesoblastic cell, and that, like such cell, it is capable of taking up foreign particles which are irritating the tissues by their presence in it.

We notice that the fate of the cell is destruction by caseation or by hyaloid degeneration, probably hastened by the metabolic products of incorporated bacilli when present. In immune animals like the ziesel, the cells do not degenerate, because the antitoxic and antibiotic influence of the blood and body juices are such that the bacilli are all destroyed before gaining entrance into the cells. If these bacilli are dead, the very existence of the giant cells is simply due to their presence as foreign particles in the tissue.

Conclusions. 1. Giant cells are cellular monstrosities, accidental and not purposeful, resulting from the overgrowth of epithelioid cells.

2. This overgrowth is brought about by mechanical or chemical irritation caused by the presence of a foreign body, whether a living or dead micro-organism or some inert particle.

3. Being the offspring of a cell possessed of amœboid and phagocytic properties, the giant cell possesses them to some degree, as shown by the ingestion of foreign particles.

4. Whether living, active tubercle bacilli are thus devoured must remain *sub judice.*

5. The giant cell exerts no special deleterious influence upon the tubercle bacillus, but seems instead to be injured by it, a more observable degeneration occurring in those cells which contain many bacilli than in those which contain none.

6. The giant cell of tubercle does not differ essentially from giant cells elsewhere, except that it shows a more marked tendency to undergo cheesy degeneration, a fact probably due to the peculiar poisonous products of the bacilli in the tubercule. *October* 13, 1892.

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11. *Cystic degeneration of the heart, spleen, liver, and kidneys.*

Read by Dr. Arthur V. Meigs.

The history of the patient is as follows: He was aged 77 years, and was born in England, but had been fifty years in America. By trade he was a printer, and had stopped work only six weeks before coming

to the Pennsylvania Hospital. There was obtained no history of hereditary disease in his family. He denied having had syphilis, but said he had drunk pretty freely all his life. Ten years before coming to the hospital he had had much bleeding from the nose, and for several years dyspnœa on going up stairs, and had passed very little urine. For six weeks he was ill, having had vomiting, aching all over the body, very infrequent micturition, and dyspnœa. He said his feet had never swollen. The attack was at first supposed to be sunstroke; for when it came on, six weeks before his admission to the hospital, the weather was excessively hot, and he was taken sick on the street. He came to the hospital August 8, 1892, exceedingly ill. By the 12th he was much worse, dyspnœa and hebetude having increased until they were excessive. August 13th, there was retention of urine, and forty ounces were drawn with the catheter. The urine contained no albumin, but there was a decided urinous odor of the body. There was bloody expectoration, and the stools were bloody. August 14th, as no urine was passed, the catheter was again used, but the bladder was found to be empty, though blood was passed from the urethra, and there was pain over the bladder. There was on this day very profuse diarrhœa, the stools being bloody. Later, he died.

No careful physical examination was made while he was in the hospital, as he was at all times too ill to be disturbed. The temperature was always subnormal, except that for a short time on the 11th it was as high as 99.4°

Post-mortem examination was made fourteen hours after death.

Lungs. The pleural sacs were obliterated by adhesions, and the lungs themselves intensely injected.

Heart. The pericardial sac was also obliterated by adhesions. The walls of the heart were thin and flabby, and the cavities dilated. The valves were natural. The arteries were in places calcareous, and there was some chalky deposit in the diaphragm.

Liver was enlarged, hardened, dark in color, and firmly adherent to the surrounding parts.

Spleen was enlarged, and firmly adherent.

Kidneys were buried in dense fat. Externally they looked about normal in size, but on section it became apparent that they were reduced by about one-third, the central spaces being filled with masses of fat. There were numerous small cysts and two quite large ones. The surface was rough and the capsule somewhat adherent.

Bladder was thickened, and contained a small amount of bloody urine.

Pieces of the heart, liver, spleen, and kidney were placed in 70 per cent. alcohol for microscopical examination.

Unfortunately, I was unable to be present at the time the post-mortem examination was made, and nothing was noticed at that time beyond what has already been described. When I came, however, to examine the pieces of heart, liver, spleen, and kidney which had been saved for me for microscopical examination, a very unusual appearance became at once evident. There were pieces from one to two inches across of each of the four previously mentioned organs. On section in any direction they are seen to be absolutely riddled with cavities, a surface three-quarters of an inch square containing quite commonly as many as fifty. Some of the cavities are as much as one-eighth of an inch in diameter. and from this size downward to the very smallest. In the liver, spleen, and kidney the cavities are spheroidal, or irregularly so at least, while in the heart the shapes are very variable, being often two or three times larger in one diameter than in another. This does not seem at all surprising when the nature of the heart tissue is remembered, the muscular fibres running in parallel bands, so that if any force was brought to bear to tear them, the fibres would separate from each other much more easily than break across their length. The cavities were all filled with liquid, which to the naked eye appeared to be clear.

These cavities in all four of the organs have the ordinary appearances of minute cysts, none of them appearing to have anything in the most distant way resembling solid contents. In many places the cysts are so close to each other that they are separated only by a thin membranous wall, and again there are openings from one to another.

Sections were cut of the organs for microscopical examination. The muscular fibres of the heart are rather opaque-looking, and the cross striæ are hardly distinguishable. The fibres are separated more widely from each other than is natural, and there is very decided increase of the intermuscular connective tissue. The fibrous or connective tissue increase is especially marked near the endocardium and around the portions of papillary muscle in the sections. Around the papillary muscles there is also some fatty tissue. Near the endocardium and around the papillary muscles there are a great many brightly stained connective-tissue nuclei. The cysts at most portions of their circumference are directly surrounded by the muscular tissue of the heart, there not being in most places a differentiated cyst wall, and in many places the muscular fibres are not even more densely pressed together around the cysts than elsewhere. Careful examination, however, brings to light the fact that in some places there is a most distinct cyst wall, quite dif-

ferent in its constitution from the muscles. In positions where this has been cut through perpendicularly so as to display it in the sections to the greatest advantage for study, it presents appearances precisely similar to those of the capillaries of the heart which I described a year or two ago.[1]

There is a fine structureless or fibrous membrane, the so-called basement membrane, and in it, here and there, small somewhat flattish nuclei, like the common endothelial nuclei of capillaries and of the intima of arteries. This differentiated cyst wall is much more commonly distinguishable in small cysts than in the larger ones; and if it is seen in those of large size, it is usually only small bits of it, thus giving the impression that the membranous walls had been torn through and destroyed as the cavities grew larger from the increase of the quantity of liquid contents. In places there is marked condensation of the muscular tissue around the cysts, and in many of the sections, here and there, portions of cyst contents have been caught, this consisting of a grumous structureless material, hardly taking the carmine stain. The amount of cyst contents must have been very small, a little flaky material hanging or floating in the liquid, hardly sufficient to make the liquid turbid, much less to give it the slightest appearance of solidity.

The liver cells are shrunken, distorted, and granular, none of them being natural in appearance. In many places only fibrous tissue is visible, but the appearance of such areas suggests rather that the liver cells had degenerated and disappeared, than that the amount of fibrous material was increased. The appearance is much like what may be seen in abscess of the liver when the secreting cells have rapidly broken down and disappeared, leaving behind only the fibrous network, and even it being misshapen from being pressed together. The bloodvessels are thickened, the branches of the hepatic artery especially being in places so thick as to be almost solid cords, there remaining only a very small opening. The bile ducts are not at all enlarged. The epithelial layer in some of them is still present, and outside of it the ducts consist of dense and thickened fibrous material. The cysts are principally surrounded by liver tissue; and as throughout the whole organ the secreting cells are small, and the amount of connective tissue visible great, this holds good around the cysts as elsewhere, and the fibrous framework as well as secreting cells are visible at the periphery of the larger cysts. In some of the larger cysts quite dense fibrous material may be seen at their

[1] "The Microscopical Anatomy of the Human Heart," Trans. of the Col. of Phys. of Philadelphia, April, 1891, and The Amer. Journ. of the Med. Sciences, June, 1891.

periphery, and it is impossible to say whence this fibrous tissue had its origin. It frequently happens that many cysts of smaller size may be seen close to each other, and these are generally surrounded by a cyst wall of fibrous material, broken through in places, the cysts thus often communicating with each other. A distinguishable cyst wall is generally the rule in small cysts, and on the other hand generally absent in large ones. Flattened cells are often visible in these membranous partitions, but they are quite large, and by their appearance suggest rather that they are altered and distorted liver cells than connective-tissue cells.

The spleen shows great thickening of the capsule, and in places the remains of the inflammatory adhesions to the surrounding parts are plainly visible. The trabeculæ are thick, and so are the bloodvessels. In places the thickening of the bloodvessels is so great that the calibre of some of them is almost occluded, and the walls have a homogeneous appearance, the differentiation of the coats being lost. The cysts are in many places surrounded by a definite differentiated membranous wall, and again there is none. The presence of a wall is more common in the smaller cysts. The cyst walls have much the same appearances as have already been described as present in the heart.

The kidney exhibits a high degree of contraction, no portion of the sections containing any normal tissue. There is great increase of connective tissue, the cells are in various stages of extreme degeneration, and the bloodvessels are fibrous and thickened, and their calibres reduced. The cysts present the ordinary characteristics of renal cysts. Sometimes there is a differentiated cyst wall, but more commonly there is not, the renal tissue constituting the edges of the cavities. The presence of a specialized wall is much more common in small than in large cavities. Structureless cyst contents are often visible in the sections, and again loose festoons hang from the walls into the cavities. It looks in many places as if some of the smaller cavities were cysts commencing in the Malpighian capsules. The capsule of the kidney is moderately thickened, and upon its surface can be seen the remains of fatty tissue in which it was imbedded, and to which it was fastened by adhesions.

Remarks.—The presence of cavities such as have been described in the four important organs—heart, liver, spleen, and kidneys—must certainly be a very rare occurrence. In looking for a cause of the condition, the first things that suggest themselves to the mind are: Could the change be a post-mortem one, or is it trichinosis? That it should be trichinosis seems quite impossible, for a careful examination of the pieces of the organs which were retained, both the naked-eye appearances and of sections with the microscope, fails to reveal anything resembling solid

bodies in any of the cavities, much less anything that could be taken to be the parasites or remnants of them. That the cavities could be the result of changes which had taken place after death, is emphatically negatived by their appearance, which is that of ordinary cysts, and by the fact that conditions of disease were present in an unusual degree of development which are common in cystic degeneration, or certainly in cases of cystic kidney. The kidneys were, as has been stated in the history, extremely contracted, and the heart fibroid, besides there being adhesion of all four of the organs to all the surrounding parts, the adhesive inflammation of the pleura and pericardium having been so extensive as to cause entire obliteration of their cavities.

It is much to be regretted that the post-mortem examination was not more thorough, for then search might have been made to ascertain if there was a parallel cystic condition of any of the other organs or tissues. If careful examination of the voluntary muscles had been made, and no evidences of disease found, this would have constituted a complete answer to anyone who might say the disease was trichinosis.

To my mind, as a result of study of the case, has come the conclusion that the cavities in the organs are cysts. Cysts in the kidney are very common, and in the liver not very rare, but of cystic disease of either the spleen or heart I have not as yet been able to find any account. It is highly improbable that in a case of cystic degeneration of four of the most important organs in the whole economy they had different modes of origin, or are in any way different from each other, further than that they are in different sorts of tissue. That cause, therefore, which produced them in one organ, probably produced them in all. With regard to the ordinary cysts of contracted kidney, there seems to be no doubt amongst pathological authorities that they always arise either in the Malpighian capsules or in the uriniferous tubules, and it is very generally accepted that cysts in the liver originate in the bile ducts. Cysts are classified in two great divisions—true cysts, and pseudo-cysts or spurious cysts. The latter are those which arise in the substance of the organs or tissues as the result of hemorrhage or disintegration, or other pathological process, while true cysts have their origin in the abnormal dilatation of a pre-existing tubule or cavity. It does not seem likely that the cavities which have been exhibited are spurious cysts, for such an assumption is negatived both by their appearance and by the fact that cysts precisely like these are so well known to occur in the kidneys and liver, which, so far as the kidneys are concerned, certainly are universally conceded not to have had that mode of origin. It is probable, as has already been said, that the cavities in the four organs had a common

mode of origin; but so far as the spleen and heart are concerned, there are neither pre-existing tubules nor cavities which could undergo abnormal dilatation except the bloodvessels and lymphatics.

My endeavor in what has been said has been to lead up to the enunciation of these two propositions—first, that true cysts do sometimes have their origin in capillaries; and second, that the commonly accepted explanation of the mode of origin of renal and hepatic cysts does not rest upon any tangible evidence which is accessible to the ordinary pathologist, and that therefore it is at least open to doubt.

So far as the first proposition is concerned, it may be well for me to say that this case has been of great interest to me in that it has seemed to furnish more evidence toward proving the assertion I made a year ago[1]—that certain cavities in the muscular fibres of the heart, the presence of which I pointed out, were cystic in their nature, that they were true cysts; and further, that they had their origin in the capillaries that I had described as lying within the muscular fibres. If it be conceded that these cavities are true, and not spurious cysts, and that such could only originate in the spleen and heart in either the bloodvessels or lymphatics, a long step would seem to have been taken toward the conclusion of their having originated in the bloodvessels.

The origin of cysts in lymph vessels is a thing as yet almost unknown; and, on the other hand, dilatations of bloodvessels, arteries, veins, and capillaries, forming aneurism, varix, or capillary aneurism, is a most common pathological appearance. For my own part, I am unable to see why vascular tumors of either of the three sorts should be classified, as is commonly done, among the spurious cysts, for it is more easy to be sure that their origin was in a pre-existing duct than in the case of almost any of the cysts which are ordinarily classed as true cysts. Whatever may be the condition of the inner wall of the vascular tumor as a result of the stretching force which is exerted from within, there can be no manner of doubt of the place in which it originated. If, then, it is so well known that cysts do originate in bloodvessels, and we know but little of their origin in lymphatics, does it not seem just to assert that the cavities that have been shown to be present in the spleen and heart are true cysts, and that their origin was within the bloodvessels?

With regard to the second proposition, my mind has long been disturbed by a feeling of dissatisfaction with the ordinarily accepted explanation of the mode of origin of renal cysts—that they arise in the

[1] "Cystic Degeneration of the Muscular Fibres of the Heart," by Arthur V. Meigs, Trans. of the Coll. of Phys. of Philadelphia, 1892, and Amer. Journ. of the Med. Sciences, May, 1892.

Malpighian capsules or in the uriniferous tubules. That they arise in the uriniferous tubules, of course, may be the case, but I have never been able to find any evidence which would go to show that such is the case. So far as their originating in the Malpighian capsules is concerned, it would seem to be borne out by the fact that often in cases of contracted kidney (and it was so in this case) the Malpighian bodies can be seen in various stages of degeneration and destruction, and the capsules dilated; but there is no more reason for supposing that this dilatation is due to obstruction to the uriniferous outlet than that disease of the capillary loop had occurred, and the dilatation had its origin within the blood-channel. What reason is there for saying that cysts in the kidney originated in the secreting structure, any more than that they may have had their origin in the bloodvessels?

December 8, 1892.

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12. *Injected tongue of a cat, showing encysted Trichina spiralis.*

Exhibited by Dr. Henry W. Cattell.

Mr. J. Percy Moore, of the Biological Department of the University of Pennsylvania, noticed, after injecting a cat, that there was some calcareous infiltration in the neighborhood of the frænum of the tongue, and gave me the specimen for study, with the result that encysted trichinæ were found in the sections. In the slide under the microscope you can see the entwining, beautifully injected capillaries around the fibrous capsule, while the ends of some show evidences of calcification.

March 23, 1893.

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13. *General tuberculosis.*

Exhibited by Dr. Alfred Hand, Jr.

On the first day of December, 1892, a boy, aged 3 years, was admitted to the Children's Hospital with a history of great thirst and polyuria, which had begun suddenly eight weeks before.

He had had entero-colitis at five months of age, and croup and measles together at two years.

The family history was negative, the parents, three brothers, and two

sisters, being alive and well. One younger sister died of entero-colitis. There had been no miscarriage.

The examination showed the boy to be undersized and puny, with light hair, brown eyes, and a bronzed, dry skin. The eyes were in a condition of exophthalmos, giving him a frog-like appearance, and there was a corneal opacity on each eye, with anterior synechiæ in the right. The thyroid was not enlarged. His chest was very small and narrow, and the rhachitic rosary could be felt; the lungs and heart were normal. His belly was distended and tympanitic, obscuring for several days the great enlargement of the liver and spleen, which at first were thought to be normal in size; there was an umbilical hernia, and the skin of the abdomen was covered with petechiæ and an eruption something like scabies. The feet were œdematous.

The urine had the following characteristics: acid, cloudy; specific gravity, 1000; quantity in twenty-four hours, 114 ounces; no sugar; albumin distinct.

The boy seemed to suffer so much from thirst that water and milk were not withheld, and he was given a drink whenever he asked for it. He was at once put upon two minims of the fluid extract of ergot, three times daily. This had no effect whatever; at the end of a week the daily amount of food ingested being 180 ounces, of which 150 ounces passed through the kidneys without taking up either albumin or sugar, the specific gravity not rising above 1000, but the color being like that of a saturated solution of picric acid. During this week the temperature had been rather irregular, but there was no decided fever until the seventh day, when the thermometer registered 102.2°. The ergot was now replaced by laudanum, two drops every six hours. Before this the thirst had not allowed more than a few minutes' sleep at a time, but the boy now began to take longer naps, and the amount of fluid taken and excreted fell to 100 ounces. During the next week the urine contained albumin once, but at the end of that time was free from it, had changed to pale yellow in color, and had a specific gravity of 1010. After this the total amount could not be collected, so an idea of the amount voided could only be gained by measuring the quantity of water and milk drunk, which decreased to about 80 ounces.

December 20th, the urine had its highest specific gravity, 1012, but the next day it fell to 1004. On the 22d the temperature rose from 99° in the morning to 105° at 4 P.M. It was normal on the 23d, and at 6 A.M. of the 24th, but at about noon it was 103°. The opium was then stopped, and one grain of quinine given three times a day, with a very beneficial result. The temperature for six weeks did not vary a degree

from normal, the liver and spleen decreased in size, the petechiæ on the abdomen gradually faded, the boy was able to get up and walk, and he ate and drank no more than a child of three years should.

February 12th, at 6 P.M., the temperature was 99°; four hours later it had risen five degrees. After a few days a broncho-pneumonia was discovered, and a further diagnosis was made, from a general view of the case, of tuberculosis. For a week the temperature was very irregular, and there was marked systemic depression. On the 19th the purpuric spots increased in number, and an elevated, dull-red macular rash appeared all over the body and extremities. At 8.30 P.M. he sat up and asked for a drink. Two hours later the body was cold, and rigor mortis had set in.

The *autopsy* was made fifteen hours after death. The rash had faded from the left side, but still persisted on the right. As the scalp was stripped off a yellow spot about the size of a five-cent piece was noticed near the right parietal eminence. When the skull-cap was removed this spot was seen on the inner side as well, and the entire thickness of the bone there was soft and movable. Other smaller areas not extending through the vault were seen on the outer surface. In the absence of any history of syphilis this was thought to be tuberculous. The brain was normal to macroscopic examination. The lymphatic glands in the omentum, mesentery, mediastina, and, in fact, all through the body, were greatly enlarged. There were a broncho-pneumonia of both lungs, a warty endocarditis of the mitral valves, and a caseous pleuritic adhesion at the base of the right lung. The liver and spleen were enlarged and firm, and the former had minute gray nodules in its substance. The pancreas was apparently normal, and there were no ulcers or scars in the intestines. The kidneys seemed slightly enlarged; the left contained three cysts, and in the pelvis of each was a yellow, hard, tubercular mass, almost filling it. Microscopical sections prepared by Dr. W. S. Carter showed nodular masses of small, round-celled infiltratration in the liver, spleen, kidneys (pelvis); a broncho-pneumonia in the lungs, and a degeneration of the epithelium of the uriniferous tubules.

March 23, 1893.

14. *Some remarks on arsenical poisoning, with special reference to its domestic sources.*

Read at the Conversational Meeting, April 27, 1893,
by Dr. FREDERICK C. SHATTUCK, of Boston.

Arsenical poisoning may be either acute or chronic. Speaking broadly, the manifestations of the acute form vary according to the mode in which the poison influences or enters the system; whether its action is mainly local on the skin or exposed mucous membranes; whether it is taken into the stomach, or reaches in a gaseous form the blood and tissues by inhalation through the lungs. We thus have inflammation of the skin or superficial mucous membranes, intense gastro-intestinal irritation, or symptoms of a very grave but more general character. There is less close relation between the manner of exposure and the character of the leading symptoms in the chronic forms, which include most cases of so-called domestic poisoning; but in these, as we shall see later, all avenues of entrance into the system may be open at once. It is interesting to note that patients who survive the early stages of acute poisoning may later, through absorption into the blood and deposition in the tissues, present symptoms identical with those which may also follow the slow and more or less continuous absorption of far smaller, perhaps of very small doses. For instance, in a husband and wife who nearly died from arsenic administered once or at most twice in their food by a daughter, who hoped thus to secure an inheritance and render herself more acceptable to a lagging lover, I have seen the development of extreme general neuritis and of pigmentation of the skin resembling, except in its universality, that of Addison's disease. In the husband ninety-three days elapsed before Prof. E. S. Wood failed to detect arsenic in the urine.[1] Such cases form part of the evidence that the metal is stored up in the system and forms albuminoid compounds, from which it is only gradually set free, and they also illustrate the double toxic action of arsenic, its local irritant and its general systemic effects. It may be mentioned that arsenic seems to be stored up in the brain and spinal cord more largely even than in the liver.[2]

The leading statements which follow admit, I suppose, of no dispute.

[1] The husband is still, after a year's interval, a paralytic and in the poor-house, I am told.

[2] Poncy and Livon: Journal de Pharmacologie et Chemie, October, 1879, p. 344. Scolosoboff: Bulletin de la Société de Chemie de Paris, xxiv., No. 3. Gautier: Annales d'Hygiène, January, 1876. p. 136.

A. 1. The continued administration of arsenic by the stomach is liable to lead to symptoms of poisoning. These symptoms may be direct—gastro-intestinal irritation; or indirect—anæmia and debility, dermatitis of different forms, skin-pigmentation, redness of the conjunctiva, puffiness under the eyes, headache, irritation of the upper air-passages, irritation of the kidneys as shown by small traces of albumin, casts, and blood in the urine, and peripheral neuritis. This list could be made longer, but comprises the more frequent manifestations. In some respects the neuritis is one of the most interesting of these. With regard to it I recall reading, but cannot now remember where, the statement of Seguin, that in his very large experience he has never seen neuritis follow the medicinal use of arsenic. In my more limited experience I have seen at least two such cases. One was that of a young woman successfully treated for chorea with large doses of Fowler's solution, which were well borne as far as the digestive organs were concerned, but she developed a marked and widespread paralysis of both motion and sensation from which recovery was slow. The other was also in a young woman, treated by me last autumn for leukæmia with Fowler's solution, gradually increased to fourteen drops thrice daily, and maintained for some weeks at that level. She developed a mild but unquestionable neuritis, and also the characteristic brown discoloration of the skin. One post-choreic case of arsenical neuritis has been observed at the Boston Children's Hospital.

2. Medical literature contains reports of a fair number of cases of poisoning by arseniuretted hydrogen, although Professor Chandler[1] says: "I never knew a case except those mentioned in the books many years ago—Galen, for instance, who put his nose to it and is said to have been killed by it—and I have never heard of one." These cases are chiefly acute and mostly rapidly fatal. The possibility that chronic non-fatal cases of poisoning originating in this way may be pretty common will be touched upon later.

3. Arsenical stockings, veils, and other articles which are liable to be closely applied to the human skin, have often been held responsible for toxic symptoms—general from absorption of the drug, as well as local from its irritant effect. A well-known Boston physician had two attacks of sore fingers. He consulted Dr. J. C. White, who asked him whether he had anything to do with arsenic. He could think of nothing except some playing cards which he had used. These were found to be loaded with arsenic, were discarded, and he has not had any similar trouble

[1] Appendix to No. 417, House of Representatives, Massachusetts, 1886.

since. Reports of general poisoning from the use of arsenical pastes, lotions, and ointments are also to be found. Dr. Stillé cites a number in his work on *Therapeutics and Materia Medica.*

B. The great difference between different persons in susceptibility to arsenical poisoning must be admitted; but the influence of idiosyncrasy does not seem to be greater here than in the case of lead, alcohol, tobacco, and many other toxic agents. It is highly probable that habituation also plays a part. Here we naturally think of the Styrian peasants so often brought up in this connection. It seems to be a fact that some of them gradually acquire the power of taking, without obvious inconvenience, doses which would prove fatal to virgin soil—six grains, for instance, it is said. But no modern physician, so far as I know, has in very recent times carefully investigated this peasantry to ascertain how many try to become arsenic-eaters and fail in their attempt; how many develop neuritis or other indisputable toxic symptoms. I cannot regard the statement of Marik[1] as conclusive. He bases his opinion that arsenical paralysis is rare in Styria upon conversations with physicians practising in that province, and on a paper of B. Knapp,[2] written in 1885, when we knew much less about neuritis, especially in its milder forms, than we do now. A degree of neuritis might well pass unnoticed in a peasant, which an artisan, for instance, would find forced upon his attention.

We have all seen patients to whom we wished to give arsenic for anæmia, chorea, or other cause, show intolerance even of the smallest doses given with every precaution. I remember a leukæmic boy in my hospital ward, who bled at the gums and grew worse in every way whenever an arsenical course was instituted, even though only one drop of Fowler's solution thrice daily was given.

C. Arsenic is widespread in civilized countries. It enters as component or impurity into many articles of domestic use. I will not weary you with an attempt to form a complete list; but among these articles may be mentioned wall-papers; cretonnes and other prints; red-striped bed-ticking; Turkey-red cotton; children's paints; the "plated" or "glazed" papers used for kindergartens, for covering paper boxes or wrapping confectionery; some woollen dress goods, hangings, and curtains; black and other stockings; some distemper paints—indeed, anything in which an arsenical aniline pigment is used as a dye. These dyes may contain from 2 per cent. to 3 per cent. of arsenic by weight. Formerly arsenic acid was largely used as an oxidizing agent in the manufacture of fuchsin

[1] "Ueber Arsenlähmungen," Wien. klin. Wochenschr., 1891, Nos. 31–40.
[2] Erg. Hefte zum Centralblatt. f. allg. Gesundheitspflege, 1885.

or magenta, but chromic acid answers equally well. Sodium arsenite is also used as a mordant to fix colors which may be non-arsenical. Silks are rarely arsenical.

D. From these articles or from some other sources in our surroundings, arsenic is absorbed into the system of many persons, as is shown by its detection in the urine of at least 30 per cent. of persons taken at random by J. J. Putnam.[1]

We now come to the question as to whether arsenical poisoning arises from the afore-named domestic sources, or, rather, whether it so arises frequently enough to demand the serious consideration of physicians, the guardians of the health of the public, as well as that of their individual patients. The burden of proof certainly rests on those who maintain the affirmative. Let us, therefore, examine the evidence for and against, beginning, as is I think usual in judicial inquiries, with the case for the plaintiff.

Readily-accessible medical literature contains the reports of a large number of cases, many of which came under the observation of highly-trained physicians presumably alive to the dangers of incomplete observation and hasty deduction, and these cases presented symptoms of varying intensity and detail, persisting in spite of what seemed judicious treatment. These symptoms were mainly subjective in some, but usually objective as well, ranging all the way from anæmia and debility to extensive peripheral neuritis. For the symptoms, no obvious cause could be discovered in the patients' manner of life, habits, or surroundings. In not a few cases a marked improvement took place on change in locality, perhaps trifling in character, while the symptoms speedily returned on resumption of home-life, without clearer assignable cause than at their first appearance. It was then found that the wall-paper of a room in which the patient slept or passed much time, the Turkey-red lining of a bed-quilt under which he slept, the red-striped bed-ticking covering his pillows or mattress, the chintz coverings of a sofa or of chairs, contained considerable quantities of arsenic. Then arsenic was also found in the urine of the patient, although it had not been given him as a drug or entered his system in Vichy or other arsenical mineral water. The kidneys were in an irritated condition. The arsenically-contaminated material was then removed and replaced by one free from the metal. This was followed by the more or less prompt abatement and disappearance of all symptoms and by restoration to health. In comparatively few of these cases is the history completed by find-

[1] Boston Med. and Surg. Journ., 1890, cxxii. p. 421.

ing the urine non-arsenical after the lapse of some months. If the patient is well, he often passes out of the observation of the physician, who may not care to subject the patient or himself to the expense of an examination which, from the point of view of the layman, seems unnecessary.

All of the reported cases are not so complete as those just sketched. In many the evidence consists in a certain grouping of symptoms for which it is difficult to account, arsenical surroundings and urine, removal of arsenic from the surroundings, and recovery. I speak of a certain grouping of symptoms. There is no one symptom which is invariably present in cases of supposed arsenical poisoning. We cannot look for a pathognomonic sign here more than elsewhere; but anæmia, debility, loss of appetite, frontal headache, perhaps nausea and vomiting, redness of the eyelids, and catarrh of the air-passages or of the intestinal tract, may be so combined as to be highly suggestive that arsenic will be found in the surroundings and also in the system.

If cases like these were isolated, occurred only in one locality, or only in the practice of one physician, one would not attach the same importance to them which one is led to attach when they are seen to occur in Great Britain and the Continental countries, in Maine and Michigan and other States as well as in Massachusetts. In reasoning from cause to effect or from effect to cause, we must often ask ourselves whether the association of results with supposed cause is merely coincidental or otherwise. I think I am alive to the ease with which, especially when public agitation is going on, members of our profession, as well as the laity, may jump at conclusions and connect things which do not belong together. The removal of a supposed injurious influence may well act as a species of mind-cure, and the reference of more or less vague symptoms of ill-health to a definite and removable cause may satisfy the patient and the physician alike. Some of the reported cases of domestic arsenical poisoning are certainly inconclusive, and I have no doubt that many persons in Boston and vicinity, particularly, have erroneously believed themselves or have been believed to be victims of this kind of poisoning. Some of the cases which have been reported and observed suggest that arsenic may be but one of two or more causes tending to produce ill-health, and that a person already debilitated from some other cause or causes may then be more suceptible to the supposed injurious influence of this poison. Prof. E. S. Wood[1] refers to the case of a young athlete whose convalescence from influenza seemed to his physician, a

[1] Hearing before Committee of Massachusetts Legislature, 1891, p. 83.

thoroughly competent man, unreasonably delayed. Arsenic was found in his urine, although the papers, carpets, etc., were free. Eighteen stuffed birds were finally removed from his room and recovery was then rapid.

Allowing full weight to inconclusive cases, and also to all other possible sources of error, it seems to me that there remains enough to make us hesitate, to say the least, before we attribute the whole thing to mere coincidence. The temptation is great to cite cases, but I shall confine myself to a series reported by C. P. Putnam,[1] and a few of my own which have not been published. When Dr. Putnam went on duty at the Massachusetts Infant Asylum in the spring of 1890, he found the babies looking very pale, without any apparent reason. He then noticed suppurations on the fingers and a slight discharge from the ears. Occasionally a nurse had sores on the fingers. One had an aural discharge, and other skin-eruptions were noticed. Arsenical poisoning was thought of as possible, but there was no wall-paper, no carpets, or other suspicious articles. The health of the inmates of the asylum grew worse. Bronchitis appeared, and two children died from inflammation and ulceration of the air-passages, involving the pleura.[2] Some blue dresses recently furnished by the asylum to all the nurses fell under suspicion and were found to contain much arsenic. They were discarded and the health of the institution was re-established. Much of the arsenic was found to be loose in the cloth. The dresses were therefore thoroughly washed in the winter of 1891 and resumed by the nurses. Very soon afterward both nurses and babies began to have sores on their fingers and other signs similar to those which had appeared before. The dresses were again discarded. The symptoms again disappeared and have not recurred in the past two years. The evidence in my own cases is not as striking, but they serve as fair illustrations of many cases seen by many physicians.

1. W., a clerk in a large dry-goods house, entered my service in the Massachusetts Hospital in the fall of 1891, with poor general health and marked neuritis in all the extremities. It was with much difficulty that he could pick a pin up from the table. Neither alcohol, rheumatism, lead, nor gout could be considered as causative of the neuritis. The

[1] Ibid., p. 66.

[2] This statement does not quite correspond with that printed in the report, but is in accordance with information given me recently by Dr. Putnam. In default of a bacteriologic examination of the lungs in the two fatal cases, it must remain an open question as to the part, if any, played by arsenic in the production of the fatal lung-disorder. The other symptoms, however, seem less open to doubtful interpretation.

physician under whose care he had been, a gentleman well known to me, had given him no arsenic. The bedroom wall-paper, which had been applied for eight years, was highly arsenical, and arsenic was found in his urine. He soon began to improve, was discharged from the hospital, and after a time resumed his work, discarding the arsenical wall-paper. I saw him very recently again, and found his general condition still below par, with some neuritis still present, though not enough to seriously interfere with his work. An electric examination, which Dr. J. J. Putnam was kind enough to make, confirmed the diagnosis of neuritis. The urine still contains a considerable amount of arsenic, the source of which I have not as yet been able to trace.

2. G., a laster, entered my service January 30, 1893. Investigation showed that no arsenic had been given him before this. Besides pallor and loss of flesh, he had ataxic paralysis of all the extremities, which proved, after thorough study, to be due to peripheral neuritis, and for which the usual causes, as laid down in the books, could not be considered as responsible. I then had the urine examined by Prof. Wood for lead and arsenic. The former was absent, the latter present in large amount. I then secured some of the paper from the bedroom which the man had occupied for two years, and also samples of the leathers on which he worked. The paper was highly arsenical, as was also one of the samples of leather. After entrance to the hospital he grew steadily worse for some weeks, a continuance of the downward tendency present when he first came under observation, and was confined to bed; but he is now greatly improved and steadily gaining in all respects.[1]

3. Two vigorous boys, brothers, had been confined to the house in town by colds and chicken-pox shortly after their return from the seashore. From these diseases they had just recovered when they, and also another brother who had been previously well, began to have recurrent attacks of nausea and vomiting. I presently suspected that some kind of poison from without was at work, and had the plumbing, cooking utensils, refrigerators, etc., examined, without finding here any cause for the illness. The symptoms grew worse. The youngest boy became extremely weak. I then had the wall-paper examined for arsenic, which was found in large quantity in that of the hall, entries, and stairway on every story, and also in that of five chambers. The papers had been on for a number of years. There was *no* arsenic in the paper on the walls of the rooms occupied by the sick children; but I found that they

[1] Repeated examinations of the urine have been made for arsenic by Wood. The amount diminished gradually, and April 24th the arsenic had practically disappeared, eighty-four days after removal from arsenical surroundings.

always slept with their windows shut and the doors into the entry open. The boys were at once removed from the house, and the papers were replaced by some free from arsenic. The patients rapidly recovered, and during the three years which have since elapsed have never had any recurrence of such symptoms. The urine of one of the boys has been examined for me within a fortnight and found to be free from arsenic. My supposition is that as the entry papers became worn, the arsenic was gradually set free in larger quantities and contaminated the air of their bedrooms, which were constantly in free and uninterrupted communication with the entry.

In none of these cases can the arsenical origin of the symptoms be regarded as absolutely proved; but I should like to call attention to the parallelism between the symptoms and those which are described in the text-books as occurring from contact with or ingestion of the drug. A few such cases may be only suggestive; but when they are multiplied manifold, as can easily be done by anyone who cares to consult the literature, the evidence furnished by them seems to me to be such as is worthy of serious consideration.

It may be mentioned that a number of European governments recognize arsenic as a source of danger in articles of domestic use.

Let us now consider the objections which can be raised—the case for the defence. In so doing I shall say what I think can be said to meet those objections, just as I called attention to some weak points in the argument for the plaintiff.

First, there is the alleged immunity of workmen and others whose occupation brings them into close relation with arsenic. A number of wall-paper manufacturers and dealers testified before the Committee of the Massachusetts Legislature of 1891 that during the many years they had been in the business they had never known of any injurious influence being exerted on themselves or their employés from arsenic. A number of such cases are, however, reported. Draper[1] mentions several cases among those employed in wall-paper shops, while cases of poisoning from green tarlatan, artificial flowers, and among taxidermists are by no means rare.[2] E. S. Wood tells me that several workmen have stated to him that they suffer every time they remove an arsenical paper

[1] Third Report Massachusetts State Board of Health, 1872, p. 17.

[2] E. G. Cutler reports his investigation of a Lowell box-factory. Two cases of illness among the employés seem fairly attributable to the arsenical green and red papers used to cover the boxes. These contained about eight grains of arsenic to the square foot, but were heavily sized. The health of the girls in general was excellent. Much attention was given to the cleanliness and ventilation of the work-room, and other precautious were taken to prevent injury. Seventh Report Massachusetts Board of Health, 1876, p. 546.

rom the walls of a room. It is quite conceivable that workmen should not care to mention their disabilities, if not too great, to their employers, for fear of losing their places. The symptoms in these cases are essentially the same as those often seen in the consumers of arsenical goods. Moreover, the conditions are somewhat different between prolonged nightly exposure to the air of a bedroom, for instance, covered with an arsenical wall-paper, and the exposure to which a clerk in a paper-shop is subjected. The papers are there tightly rolled and stored on shelves. But small portions of the rolls are displayed to customers, and only for a short time. The shop, moreover, must be dry, or the papers would mildew and become unsalable. Arsenical dust is, therefore, the only thing to be dreaded. As far as reports in medical literature go, there certainly seems to be a relative immunity among workmen. Whether this immunity is really as great as it seems is a question which appears to me worth further study. The observance of simple precautions can, undoubtedly, do much to lessen the danger.

Next, we have the fact that far larger quantities of arsenic in solution —*i. e.*, in a condition ready for immediate absorption—can be and constantly are given medicinally by the mouth with impunity or only transitory gastro-intestinal disturbance than it is conceivable should, save in very rare instances, enter the system from domestic sources. Chemists of considerable experience in the examination of urine for arsenic recognize this fact, and I have repeatedly known the report to be made that the amount was so large that it must, in part at least, have been taken somehow by the stomach. Elimination after ingestion is slow. In a patient of mine to whom twenty-seven drops of Fowler's solution were given during three days, arsenic was detected in the urine by Wood until the fifty-eighth day. In another, who received sixty-nine drops in seven days, eighty-two days were required for elimination. But the conditions of gastric administration and exposure to arsenical papers or goods are not identical. There are reasons for believing, as will appear more fully later, that some gaseous form of arsenical compound may be set free in consequence of the growth of certain moulds, and that in this form and inhaled the poison may be more active than when it enters the system exclusively through the stomach. But whether arsenic is given off as a dust or gas, the duration of the exposure is generally quite different; that is, it is likely to be far longer in the domestic cases. In either case habituation may, but does not necessarily, ensue.

This leads us to the next objection, that arsenic is found in the urine of many persons who are quite free from indications of subjective or

objective disease. J. J. Putnam has conclusively shown that the same is true of another metal, lead, the toxic influence of which on some persons is admitted by all. If this objection is to have weight with one metal, it would seem to have equal weight with the other. Idiosyncrasy, which includes relative immunity and habituation, must relate to both or neither.

The objection that arsenical poisoning of domestic origin occurs only in Boston would be too trivial to notice here were it not that it has been seriously urged, though, it is true, mainly by interested parties. It so happens that the question has received more attention in Boston than perhaps elsewhere in the United States; but the statement that cases are not observed elsewhere argues simply the ignorance of the person making the statement. There are plenty of unrecognized relations between cause and effect, plenty of unclassified conditions which are constantly before our eyes if we could only see them. The general recognition of peripheral neuritis is a very recent matter.

The risk is considered sufficiently great to warrant legislative interference in Sweden, Germany, Russia, Austria, and Denmark. In France no regulation exists, except with reference to articles of food and drink and children's toys, though the government has sent circulars to manufacturers warning them of the penalties to which they are liable if accidents result from the presence of poisonous substances in their goods. In England there is no law, but there has been considerable agitation on the subject, especially by the National Health Society and the London *Lancet*.[1] The very agitation has, however, been followed, as it is stated, by a marked diminution in the amount of arsenic contained in articles for domestic use. An abstract of the laws on this subject will be found in the report of the Massachusetts Board of Health for 1885, in an article from the pen of E. S. Wood, entitled "Arsenic as a Domestic Poison." This abstract is taken from the English National Health Society's report for 1883.

I have tried to ascertain what changes, if any, have been made in these laws since 1885, and am indebted to Vorban[2] for most of the statements here immediately following. The Swedish laws, the most stringent and precise, remain the same since 1883. The maximum amount of arsenic permissible in wall-papers, materials for artificial flowers, and some other articles, is about one-fourteenth of a grain to the square yard; in textile stuffs and yarns and other specified articles one-seventh of a grain to the square yard. The process to be used in

[1] 1892, ii. 43.

[2] Inaugural Dissertation, Dorpat, 1889.

the chemical determination is carefully defined. The German law of 1882 was found to be too stringent in that it absolutely prohibited arsenic in wall-papers and articles of apparel. The metal is so widespread and the means for its detection are so delicate that minute traces can be found, oftentimes in spite of the best faith and most painstaking care on the part of the manufacturer. The law of 1887, which went into effect May 1, 1888, allows a maximum of two and one-half grains to the square yard, provided that the arsenic is in a form insoluble in water. An exception is also made in favor of colors which do not contain the arsenic as a constituent part, but simply as an impurity; and this at the most in a quantity unavoidable in the ordinary methods of manufacture. Obviously, then, exceptions might afford opportunities for dispute and a loophole for a dishonest manufacturer. The Chancellor of the Empire is authorized to issue directions as to the process to be used in the detection and estimation of the amount of the metal.

The Russian general law of 1876 seems to be still in force. It allows a maximum of from one-seventh to one-eighth of a grain of arsenic to the square yard in wall-papers. There are some local regulations, as in Riga and Dorpat. In the latter place the maximum is fixed at one-fifteenth of a grain per square yard in wall-paper and one-thirtieth of a grain in cloth.

In this country, Massachusetts takes the lead in the agitation on the subject. The ball was opened by Dr. Draper in 1872, and four unsuccessful attempts have been made to secure legislation. The act of 1891 prohibits arsenic in confectionery and children's toys, and authorizes the Board of Health to expend a sum not exceeding one thousand dollars on "such investigations and inquiries as they deem necessary as to the existence of arsenic in any paper, fabric, or other article offered for sale or exchange." The outcome of this is the report of Prof. W. B. Hills,[1] with regard to the conclusions of which I shall speak later.

Recognition of the danger which may exist of domestic arsenical poisoning is found in the reports of Boards of Health of Michigan for 1873, California for 1874, Rhode Island for 1878, Connecticut for 1879, Pennsylvania for 1887, New York for 1888, and Maine for 1889, as well as in those of Massachusetts for 1872, 1889, and 1892. In the year 1874 the Committee on Poisons of the Michigan State Board of Health prepared one hundred books of specimens of poisonous papers bought from first-class dealers in various cities, and placed these books in public libraries. The book was entitled *Shadows from the Walls of Death, or*

[1]Report of Massachusetts State Board of Health for 1892.

Arsenical Wall-papers. This title was followed by a quotation from Leviticus, 14th chapter: "And behold, if the plague be in the walls of the house, with hollow strakes, greenish or reddish, . . . Then the priest shall go out of the house to the door of the house, and shut up the house seven days. . . . And he shall cause the house to be scraped within round about, and they shall pour out the dust that they scrape off without the city into an unclean place." I find the plague referred to is leprosy. One lady is said to have been poisoned by examining this book.

In the report of the State Board of Health for Pennsylvania for 1887, in the report of the Committee on Adulterations, Poisons, etc., it is stated that but little work has been done during the year for lack of funds and by reason of the expensiveness of chemical investigation, although "in the manufacture of certain foods employed in our households, wall-papers, textile fabrics, dye stuffs, etc., we shall find abundant scope for the operation of the Board's conserving energies." The only State besides Massachusetts in the laws of which wall-paper is mentioned in connection with poisonous pigments, as far as I can find, is New Hampshire, and here the matter is left in such a way that it can have no direct practical result.

We see, then, that the positive evidence is strong and comes from many sources. It seems to me far stronger than the negative evidence which I have tried to present fairly. It would certainly appear to be undesirable that the public should be exposed to an influence which there is so much reason to believe to be injurious, especially when such exposure is quite unnecessary. In proof that it is unnecessary I will call only three witnesses.

Prof. W. B. Hills reports in 1892 to the State Board of Health of Massachusetts, that in the last ten years decided improvement has taken place in the wall-papers sold in the State. About 3 per cent. of the papers manufactured to-day contain more than one-tenth of a grain of arsenic to the square yard, against, approximately, 30 per cent. ten years ago. Between 60 per cent. and 70 per cent. of the papers sold in the State are free from arsenic. About 90 per cent. contain less than from one-twentieth to one-tenth of a grain per square yard, an amount which can be harmful to probably very few people. Prof. Hill states that arsenic is not essential to the production of the colors, and that those which were formerly arsenical can now be made without that metal. An absolutely exact comparison between the examinations made from 1879 to 1881 and from 1889 to 1891 is impossible, for the reason that during the latter period the so-called Marsh-Berzelius test was used, while in the earlier

period the original Marsh test was followed. The modified method is much more delicate than the Marsh test and detects quantities of arsenic which escape the latter. A still more striking improvement is shown in the "glazed" and "plated" papers used in kindergartens, book and pamphlet covers, to wrap lozenges, etc. The use of Paris green is much less common in tickets, show-cards, and the like than it was ten years ago.

Mr. Henry Saltonstall, the treasurer of the Pacific Mills, one of the largest cotton mills in the world, testified before the Legislative Committee of 1891,[1] with authority to speak for four of the six print works in Massachusetts, thus: "I wish to say in behalf of them (the print works) that not only are we not averse to a law restricting the use of arsenic, but we welcome such a law. . . . We would be glad to have a reasonable and fair law which should prohibit the use of arsenic by any mills inside the State or factories outside the State which have their goods for sale here. . . . I don't dare to say what would be the danger-limit, but I think if one-fiftieth of a grain of soluble arsenic were allowed per square yard we could come down to this limit in textile fabrics or furniture." The proposed law, which failed to pass, allowed one-twentieth of a grain per square yard, not distinguishing between the soluble and insoluble forms. This is strong testimony to show that there is no necessity for the use of dangerous quantities of arsenic, coming, as it does, from a most competent general chemist, and also from a very large manufacturer, who has presumably consulted with his own chemists before he committed himself.

Mr. Jones, student-assistant in the chemical laboratory of the Harvard Medical School, showed me an old sample of green tarlatan which contained one-half its weight of arsenic. He tells me he has bought samples recently and failed to find any arsenic in them. Until very lately many of the red-striped bed-tickings contained much arsenic, but they, too, show the effect of agitation on the subject. In connection with the preparation of this paper I meant to experiment on myself. Professor Wood kindly examined my urine and failed to find arsenic. I then tried to buy arsenical bed-ticking to cover my pillow, taking from Wood a sample which was largely impregnated. Both pieces which were bought for me proved to be non-arsenical, so I was unable to complete my experiment and find out whether my urine became arsenical after sleeping on this pillow-covering.

We are next led to the question as to what amount of arsenic is allow-

[1] Hearing before the Legislative Committee on Public Health, 1891, pp. 38 and 41.

able, a question which nobody seems prepared to answer absolutely. It is, however, probable that an amount not exceeding, say, one-fifteenth of a grain to the square yard, irrespective of solubility and form, may be regarded as practically innocuous. In the German experience we find one explanation for the difficulty in framing a law which will work no injustice to manufacturers or harm to consumers. The best tests are so delicate, and the presence of minute traces of arsenic so general in one form or another that absolute prohibition is really impracticable. A satisfactory law must, like the Swedish, not only fix the maximum allowable amount, but must also prescribe the method of chemical analysis. It is probably not practicable to distinguish, legally, between soluble and insoluble arsenic.

Some manufacturers and dealers argue that the diminution in the amount of arsenic contained in papers and fabrics during recent years shows in itself that no law is necessary. But this diminution cannot be attributed to anything except the agitation of the subject, a continuance of which demands considerable public spirit on the part of the agitators, whose ranks are not generally largely recruited from among the moneyed class. It is, moreover, doubtful whether the benefits of the agitation in Massachusetts extend widely beyond the State. Arsenical goods are, in the main, unsalable there; but no questions are asked in most parts of the country. If purchasers ask no questions, even in Massachusetts, they are liable to receive dangerous goods.

The question is interesting, and also important, as to the manner of introduction of arsenic into the system from domestic sources. When the arsenical material comes into close contact with the skin, whether broken or unbroken, as in stockings or in arsenical caustic paste, the avenue of entrance is plain. When absorption takes place from a distance, as in the case of wall-papers, the arsenic is set free either in the form of dust or in a gaseous state. In the former case it is swallowed and inhaled; in the latter, inhaled alone. There can be no question that arsenical dust is detached from papers, fabrics, water-colors, sometimes from curtains or carpets. The dust which has settled on ledges, etc., in rooms containing arsenical papers has been analyzed and found impregnated with the metal. The ease with which the poison is detached varies, of course, much with the character and surface of the paper and material. Some papers grow much more dangerous with age as they become worn; others are highly dangerous from the start. Covering an arsenical with a non-arsenical paper does not necessarily render the former innocuous. Varnishing an arsenical paper renders it safer, certainly for a time, but does not secure safety. Arsenic is sometimes

used as a preservative for the paste which fastens the paper to the wall.

I think it was Fleck[1] who, about twenty years ago, first supported by scientific evidence the suggestion which had been previously made that a gaseous form of arsenic may, with the aid of moisture and in the presence of organic substances, be set free from arsenical pigments. Hamberg[2] detected the metal in the atmosphere of rooms with arsenical papers. The nature and manner of production of this gas has been studied by Husemann,[3] Selmi,[4] and very recently by Gosio.[5] The work of the latter is not very readily accessible, and I therefore venture to abstract a portion of his conclusions.

It is proved beyond question that arsenical gases can be developed by the growth of moulds in contact with arsenical chemical compounds. Only a few moulds possess this property. While many moulds can live and grow in the presence of arsenical compounds, only four have thus far been proved to possess the power of setting free arsenical gas: mucor mucedo, aspergillus glaucus, aspergillus virens, and penicillium brevicaule (the most important).

The conditions which favor the production of arsenical gas by these moulds are, first, those which favor in general the life of the fungus, abundance of oxygen, moisture, nutritious material, and suitable dosage of arsenic. Secondly, those which depend on the facility of the transformation of the arsenic. Thus arsenic acid, and the arsenites, and arsenites of sodium and potassium are more easily transformed than the arsenite of copper. Third, the presence of carbohydrates (starch pastes). These moulds can, however, decompose arsenite of copper from papers even when they grow in the paste or glue fastening them to the wall. In the case of mucor mucedo, at least, the quantity of arseniuretted hydrogen evolved is small in comparison with another more important compound, probably the union of the metal with organic alcohol or aldehydic radicals.

These conclusions are the result of work up to May, 1892. Its further prosecution is promised. They are quite confirmatory of the English reports which suggest that arsenical wall-papers exert a more deleterious action during moist than during dry weather, and they throw important light on one method of domestic poisoning.

The main, if not the sole avenue of elimination of arsenic which has

[1] Zeitschrift f. Biologie, 1872, viii. 444; et seq.
[2] Schmidt's Jahrb., 1875, Bd. clxv. 240.
[3] Virchow-Hirsch's Jahresb., 1881, S. 413.
[4] Schmidt's Jahrb., 1875, Bd. clxviii. S. 60.
[5] Monograph, Ministry of the Interior, Scientific Laboratories of the Board of Health Rome, 1892.

got into the circulation in one way or another is the kidneys. It is a practical point worthy of note that we do not as yet know any direct means to further the elimination. The treatment of chronic arsenical poisoning resolves itself into, first, stopping further absorption; second, general hygiene; and third, the treatment of such symptoms as may require it. Active catarrhal symptoms are likely to subside pretty quickly on the removal of the toxic agent. Neuritis is to be treated by massage and electricity, just as when due to other causes.

The prognosis is good, as regards both life and recovery. Fatal cases are rare, and recovery is relatively quick, unless neuritis is extensive or very advanced, or the arsenical poisoning is only one of a combination of causes producing ill health.

The method of detection used by all the gentlemen connected with the chemical department of the Harvard Medical School is the Marsh-Berzelius test, which is sufficiently delicate and easy of application. In determining the length of time necessary for the elimination of arsenic, Wood has used the method of Sänger.[1]

The conclusions I feel justified in drawing from the foregoing considerations, which I have endeavored to strip of unnecessary detail, are the following:

1. There is overwhelming evidence to show that many arsenical articles which enter into common domestic use are dangerous to health.

2. That while it is impracticable to demand absolute freedom from arsenic in these articles, the presence of injurious quantities of the metal is quite unnecessary.

3. That, therefore, carefully framed legislative restriction, if possible uniform throughout the States, is desirable.

4. That it is the duty of the medical profession everywhere to study the question, and to lend its powerful aid in forming public opinion and otherwise securing such legislation.

I wish to take this opportunity of expressing my indebtedness to Drs. E. S. Wood and J. J. Putnam and to Dr. J. L. Morse for indispensable assistance in searching the literature.

[1] Boston Medical and Surgical Journal, April 27, 1893.

15. *Tuberculosis in a dog.*

Exhibited by Dr. Henry W. Cattell.

Tuberculosis is a rare disease in the dog, and usually results from the dog's licking up the sputum of a tubercular master. I had the pleasure of making the autopsy on the present case by the kindness of Dr. Leonard Pearson. Nodules were found widely disseminated and very numerous, especially in the thoracic cavity and the upper part of the abdominal cavity. Some of these nodules were of large size and presented the features rather of secondary carcinoma, as we see it in man, than of tubercles. The specimen which I place under the microscope is from the liver. The literature of the subject is given fully in the work of Friedberger and Fröhner.[1]

Dr. McFarland: It happened accidentally that a portion of a tuberculous nodule from this very dog fell into my hands, with the request that I would search for tubercle bacilli in it. This I did, but was unable to find any. My observations were rather carelessly done, and the absence of the tubercle bacilli does not prove that the case was not one of tuberculosis, yet I was surprised that I found no bacilli, for when present I rarely fail to detect them. The absence of the bacilli, and the fact that the animal was a dog, led me to think of the case as one of *pseudo-tuberculosis.*

In the act of inspiration we all take into our lungs a number of fine particles, in greater or less amount, according to our abode and occupation. Supposing the particles to be large, they are generally stopped in the bronchioles, driven outward by the cilia of the epithelium, eventually mixed with the mucus of these tubes, and expectorated. Smaller particles gain entrance into the air cells, where they excite more or less irritation according to their irritant properties. Like foreign particles elsewhere in the body, they excite a phagocytic action on the part of the epithelial cells of the alveoli, and also in the leukocytes. The alveolar epithelium proliferates somewhat, and these cells pick up numbers of the particles, which are finally expectorated. The leukocytes, with their burdens enter the lymphatics, and if unobstructed in their journey ultimately reach the bronchial lymphatic glands. If obstructed by any cause, as accumulation of similar cells, these leukocytes die, deposit their load of material in the lymph vessels, imposing the task of caring for these particles to the endothelial cells of the lymph

[1] Specielle Pathologie und Therapie der Hausthiere, part 2, p. 574.

vessels. In this way it is that, in cases of miners' lungs, we see so many dark lines pursuing tortuous directions beneath the pleuræ. If the particles which reach the air vesicles are larger than can be disposed of by phagocytes, or are in too large a number for these active cells to relieve the air cell, a local inflammatory process, characterized by hyperæmia, proliferation of epithelium, transudation, and some fibrin-formation is set up. As there is no way for the affected part to be freed of its irritant, the process becomes chronic, transudation and transmigration cease, and slowly and in concentric layers new connective tissue is deposited about the intruding materials, and nodules are found which somewhat resemble tubercles. I need not tell you that this form of pseudo-tuberculosis is called *pneumonoconiosis*. When particles of coal are the source of the trouble the disease is easily recognized by the color, but when glass, which is invisible, or lime, which is white, it is much more difficult to make out just what the cause of the trouble has been.

The study of inert particles has taught us that we may also inspire living entities which are capable, by continuous growth and development, of bringing about similar changes of more malignant nature, because whenever started on its career, these irritants spread from place to place, multiplying on the inside, and not needing reinforcement from without. Such a disease is tuberculosis, which is too well known to need special description. Various observers have found that when normal saliva is pulverized and sprayed into the respiratory tract of animals, not infrequently a disease similar to tuberculosis occurs in the lungs, spreading to other parts of the body and appearing in nodular form in the liver, kidney, and spleen. This disease differs from tuberculosis in that the tubercle bacillus is *constantly absent*. It is called *pseudo-tuberculosis*, and to-day when this term is used it always means a bacteridian disease, in contradistinction to pneumoconiosis. Charrin and Roger observed a case of this disease arise spontaneously in a guinea-pig, and subsequently induced the same in a rabbit, guinea-pig, and white rat by inoculation. Moreover in these cases, in which the tubercle bacillus was absent, another organism specific for the disease and denominated the *bacillus pseudo-tuberculosis* was found.

The bacillus of pseudo-tuberculosis is most easily differentiated from that of tuberculosis, for while the former will grow only on blood serum, or in agar-agar and bouillon to which considerable glycerin has been added, the latter grows readily on all ordinary media. The pseudo-bacillus also is actively motile, and will not retain stains against the action of strong acids as the tubercle bacillus does.

The tubercles of pseudo-tuberculosis show less disposition to degenerate,

fewer giant cells are found, and more well-formed connective tissue, than true tubercles. At a glance one can sometimes distinguish these tubercles from true tubercles by the fact that the centre stains as well as the periphery. My studies of this case were too limited for a positive statement, yet I am prone to regard this case in the light of pseudo rather than true tuberculosis, especially as the disease has occurred in a dog which is one of the animals peculiarly immune against tuberculosis.

May 25, 1893.

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16. *Progressive muscular atrophy.*

Exhibited by Dr. H. W. Cattell.

The specimen I exhibit is the diaphragm from a case of progressive muscular atrophy. The patient was a girl aged 17 years. She was emaciated to the last degree, being literally skin and bones. No scales was at hand, but I judge the weight was not over 26 or 27 pounds. I show the diaphragm because of its great tenuity, the muscular fibres having almost completely disappeared in certain parts.

June 8, 1893.

INDEX.

ABBOTT, A. C., the factors concerned in the production of suppuration, 248
Abortion, criminal, 207
Abscess of heart, 80
of liver, 43
of ovary, 209
Address of President, Annual, 260
Albuminuria, the, of uric acid and oxaluria, 169
Allyn, H. B, cerebro-spinal meningitis, three fatal cases of, 216
discussion, 230
osteo-sarcoma of tibia, 13
Anæmia, pernicious, heart from, 95
spinal cord from, 221
Aneurism, aortic, 79, 81, 97, 106, 109, 110, 137, 148
beginning, 100
cardiac, 102
dissecting, of aorta, 144
of basilar artery, 149
Anomalies of circle of Willis, 93, 149
of kidney, 161
Anthracosis, 246
Aorta, aneurism of, 79, 81, 97, 106, 109, 110, 137, 148
beginning, 100
dissecting, 144
rupture of, 108
Aortic valvular disease, 101
Arsenical poisoning, its domestic sources, 285
Arthritis, tubercular, 12
Ascites in peritonitis, 55
Ashhurst, John, tuberculosis of bones and joints, 1
Atheroma of aorta, 136
with beginning aneurism, 100
of heart-valves, 103
Atrophy, progressive muscular, 303
of motor speech-centre, 220

BALL, M. V., aneurism of aorta, 110
discussion, 140
peritonitis, 55
Ball, M. V., tuberculosis of lungs and serous membranes, 157
Basilar artery, aneurism of, 149
Bladder, cancer of, 166
dilatation of, 190
hypertrophy of, 167
laceration of, 17
Bloodgood, J. C., tubercular arthritis, 12
Blood, leukæmic, 90
methods of examining the, 122
pathology of the, 111
Bones, tuberculosis of, 1
Brain, globular, in epileptic insanity, 221
Breast, cancer of, 209, 212
fibroma, multiple soft, 211
sarcoma of, 210
sero-cystic, 211
Bright's disease, the albuminuria and, of uric acid and oxaluria, 169
Bronchus, rupture of lung and, 158
Brown, F. K., cancer of stomach and gall-bladder, 76
Burr, C. W., atrophy of motor speech-centre, 220
cancer of liver and gall-bladder, 42
congenital cystic kidneys, 168
discussion, 53
epithelioma of face; aspiration pneumonia, 154
globular brain in epileptic insanity, 221
spinal cord from a case of pernicious anæmia, 221
Burtenshaw, J. H., epithelioma of perineum, 226

CALCIFIED lipoma, 230
Cancer, multiple, involving cerebellum, 213
of bladder and uterus, 166
of breast, 209, 212
of gall-bladder, 42, 51, 73, 76
of liver, 42, 50, 75

Cancer of œsophagus, 72
of stomach, 47, 48, 74, 75, 76
colloid, 37, 45
of uterus, 166, 204, 209
scirrhous, of breast, 212
Carcinoma (*see* Cancer).
Cat, sarcoma of pia mater of, 215
Cattell, H. W., aneurism of basilar artery, with anomalous circle of Willis, 149
bilateral double ureters, 202
cancer of œsophagus, 72
of stomach, 74
canine tuberculosis, 301
cylindrical epithelioma of omentum, 58
cyst of kidney, 169
dissecting aneurism of entire aorta, 144
encysted empyema, 155
extra-uterine pregnancy causing volvulus, 75
hemorrhagic erosions of stomach, 23
irreducible inguinal hernia, 70
multiple soft fibromas of breast, 211
pericarditis, 144
progressive muscular atrophy, 303
tongue of cat, showing trichina spiralis, 282
Cerebellum, cancer of, 213
Cirrhosis of liver, 99
Cohen, S. Solis-, discussion, 188
enlarged heart with valvulitis, 138
gangrene of lung from disease of fibula, 155
membranous enteritis, 63
tuberculous broncho-pneumonia, 154
Colloid cancer of stomach, 37, 45
Colon, epithelioma originating in, 58
giant growth of, 23
Coplin, W. M. L., discussion, 230
Cylindroids in the urine and their significance, 191
Cyst, dermoid, of mouth, 56
hydatid, of liver, 71
of heart, 275
of kidneys, 163, 164, 168, 275
of liver, 69, 275
of ovary, 203, 209
of peritoneum, 74
of spleen, 275
of vermiform appendix, 54
Cystic degeneration of heart, spleen liver, and kidneys, 275
disease of liver, 69
elephantiasis, congenital, 241

DA COSTA, J. M., the albuminuria and Bright's disease of uric acid and oxaluria, 169
Daland, Judson, atheroma of aorta, 100
cancer of stomach, 48
dilated right ventricle, 100
Davis, G. G., exostosis (ungual), 16
fracture of elbow, 14
of pelvis, 17
of radius (Barton's), 15
scirrhus of breast, 212
traumatic rupture of lung and bronchus, 158
Deaver, J. B., discussion, 151
epithelioma of penis, 210
fracture of spine and ribs, 13
pyosalpinx, 208
sarcoma of breast in a boy, 210
Dermoid cyst of mouth, 56
Downes, A. J., tuberculous intestines, subperitoneal hernia, 61
Duodenal ulcer, 49

ELBOW, fracture of, 14
Elephantiasis, congenital cystic, 241
Ellett, E. C., colloid cancer of stomach, 37
Embolism, multiple, 100
Emphysema, pulmonary, in an infant, 141
Empyema, encysted, 155
Endocarditis, 71, 110
malignant, 98, 137, 143, 147, 223
Enteritis, membranous, 63
Epilepsy, globular brain in, 221
Epithelioma of face, 154
of omentum, 58
of penis, 210
of perineum, 226
Erosions, hemorrhagic, of stomach, 23
Eshner, A. A., aneurism of thoracic aorta, 106
cancer of gall-bladder and pylorus, 76
discussion, 67, 69, 140, 189
valvular disease of heart, 104

Exostosis, ungual, 16
Extra-uterine pregnancy, 203, 209
causing volvulus, 75

FEMUR, ostitis of, 17
Fibroma of uterus, 203, 204, 205
multiple soft, of breast, 211
Fisher, H. M., multiple sarcoma, 227
pericardial effusion and mitral stenosis, 134
suspected leprosy, specimen of skin, 240
Fistula, gastro-duodenal, 48
Flick, L., discussion, 236
Formad, H. F., abscess of heart, 80
aortic aneurism, 79
criminal abortion, 207
cystic kidneys, 163
discussion, 215, 227, 246
giant growth of colon, 23
gunshot wound of mouth, 36
hypertrophy of heart, 81
of prostate gland, 210
liver from a case of jungle fever, 44
malignant endocarditis, 98
pregnant uterus, with fœtus, 207
purpura hæmorrhagica, 96
stomach, hydrocyanic acid poisoning, 45
Fracture, Barton's, 15
of elbow, 14
of pelvis, with laceration of bladder, 17
of radius, 15
of spine and ribs, 13
Fussell, M. H., abscess of liver, 43
aneurism of aorta, rupture, 97
stricture of œsophagus in a horse, 45
typhoid fever with perforation, 20

GALL-BLADDER, cancer of, 42, 51, 73, 76
Gall-stones, 71
causing intestinal obstruction, 68
Gangrene of lung after fibular disease, 155
Gastro-intestinal tract, secondary tuberculosis of, 235
Giant-cell, the, of tubercle, 262
Gout, kidneys from a case of, 160
superficial lesions of, 240
Griffith, J. P. C., discussion, 55, 105
fatty heart from a case of pernicious anæmia, 95
Guitéras, John, secondary tuberculous lesions of the nervous system, 232
Gynecological specimens, miscellaneous, 203, 209

HAMANN, CARL, horseshoe-kidney, 199
Meckel's diverticulum, 68
tubercular or malignant disease of peritoneum, 57
Hand, Alfred, Jr., double aneurism of aorta, with rupture, 148
general tuberculosis, 282
Heart, abscess of, 80
aneurism of, 102
atheroma of, 103
bovine, 110
cysts of, 275
dilated, 165
right ventricle, 100
fatty, 95, 101
hypertrophy of, 81
and dilatation, 99, 138
in pernicious anæmia, 95
valvular disease, 104
Henry, F. P., the pathology of the blood, 111
Hernia, inguinal, 70
subperitoneal, of omentum, 61
Hodgkin's disease, specimens from, 94
Hughes, W. E., aneurism of heart, 102
discussion, 53, 61, 103, 231
hydatid cyst of liver, 71
malignant endocarditis, 143, 147
retro-peritoneal sarcoma, 230
Hydrocyanic acid poisoning, stomach in, 45
Hypospadia, 190

INTESTINES, obstruction of, 41, 68
of typhoid fever, 18
perforation, 26
tuberculous, 61

JAMISON, W. B., aneurism of aorta, 109
rupture of aorta with hæmatoma, 108
Joints, tuberculosis of, 1
Jungle fever, liver from a case of, 44

KIDNEYS, absence of one, 190
anomalous, 161
congenital cystic, 164, 168
contracted, 99, 103
cystic, 163, 169, 275
from a case of gout, 160
horseshoe-, 199
pyo-litho nephrosis of, 167, 200
stone in the, 200

LAPLACE, ERNEST, discussion, 41, 200, 227
foreign body in rectum, 54
Leidy, Jos., Jr., anomalies of circle of Willis, 93
discussion, 54
intra-uterine polypoid fibroma, 208
Leprosy, suspected, skin of, 240
Leukæmia, blood of, 90
Lipoma, with calcification, 230
Liver, abscess of, 43
atrophic, 99
cancer of, 42, 75
primary cirrhotic, 50
cystic, 69, 275
cyst of, hydatid, 71
from case of jungle fever, 44
transfixed by pin, 42
Lloyd, J. H., gall-stones, 71
multiple neuritis and malignant endocarditis, 223
perforating ulcer of stomach, 58
stone in the kidney with multiple purulent cysts, 200
Lungs, acute tuberculous broncho-pneumonia, 154
aspiration pneumonia, 154
croupous, 156
emphysema of, in an infant, 141
gangrene of, 155
lesions secondary to tuberculosis of, 232
rupture of, 158
tuberculous, 157

MAMMA (*see* Breast).
Massey, Frank, sero-cystic sarcoma of breast, 211
McFarland, Jos., discussion, 61, 301
the giant-cell of tubercle, 262
McMurtry, L. S., discussion, 205
Meckel's diverticulum, 68
Meigs, A. V., cystic degeneration of heart, spleen, liver, and kidneys, 275
discussion, 13, 105, 140, 151, 166, 189, 215, 231
president's annual address, 260
renal thrombosis and pulmonary emphysema in an infant, 141
Melanotic sarcoma of neck, 229
Membranous enteritis, 63
Meningitis, cerebro-spinal, 216
Mesenteric glands, suppurating, causing intestinal obstruction, 41
Miller, D. J. M., anthracosis, 246
cancer of stomach and liver, 75
hypertrophied heart, atrophic liver, and contracted kidneys, 99
thrombosis of heart and pulmonary artery, 136
Mitchell, J. R., colloid cancer of stomach, 45
discussion, 40
Mitral stenosis, 134
Mouth, dermoid cyst of, 56
gunshot wound of, 36
Musser, J. H., aneurism of aorta, 79
discussion, 187
secondary tuberculosis of the gastro-intestinal tract, 235
superficial lesions of gout, 240

NECK, melanotic sarcoma of, 229
Nephritis, chronic, 165
chronic interstitial, 99, 103
parenchymatous, in croupous pneumonia, 156
Neuritis, multiple, with malignant endocarditis, 223

ŒSOPHAGUS, cancer of, 72
stricture of, in horse, 45
Omentum, epithelioma of, originating in colon, 58
Osteitis (*see* Ostitis).
Osteo-sarcoma of tibia, 13
Ostitis of head of femur, 17
Ovary, abscess of, 209
cystoma of, 203, 209
Oxaluria, relations to albuminuria and Bright's disease, 169

PACKARD, F. A., discussion, 166, 220
verminous pneumonia of a Rocky Mountain sheep, 152
Papilloma of rectum, 56
Pearce, F. S., cancer of bladder and cervix uteri, 166
endocarditis, bovine heart, 110
kidneys from a case of gout, 160
liver of chicken transfixed by a pin, 42
multiple cancer involving cerebellum, 213
pseudo-leukæmia, 94
pyonephrosis and hypertrophy of bladder, 167
typhoid fever and purpura hæmorrhagica, 18
Pelvis, fracture of, 17
Penis, epithelioma of, 210
Pericardial effusion, 134
Pericarditis, acute, 144
hemorrhagic, 138
Perineum, epithelioma of, 226
Peritonitis, in typhoid fever, 20
sarcomatous or tuberculous, 55, 57
Pia mater, sarcoma of, of cat, 215
Pneumonia, aspiration, from epithelioma of face, 154
broncho-, acute tuberculous, 154
croupous, 156
thrombosis of heart and pulmonary vessels in, 136
verminous, of sheep, 152
Polyp, intra-uterine fibroid, 208
Pons, softening of, 219
Pregnancy, extra-uterine, 75, 205, 209
President's annual address, 260
Price, Jos., miscellaneous gynecological specimens, 203, 209
pyonephrosis, 200
Prostate gland, hypertrophy of, 210
Pseudo-leukæmia, 94
Purpura hæmorrhagica, 96
in typhoid fever, 18
Pyonephrosis, 167, 200
Pyosalpinx, 205, 208, 209

RADIUS, Barton's fracture of, 15
Rectum, foreign body in, 54
papilloma of, 56
Renal thrombosis in an infant, 141
Retroperitoneal sarcoma, 230
Ribs, fracture of, 13
Richards, R. M., anomaly of kidney, 161
Ross, George, pyosalpinx, 208

SARCOMA, melanotic, of neck, 229
multiple, 227
of breast, 210
of pia mater of cat, 215
of tibia, 13
of uterus, 204
retroperitoneal, 230
Scirrhus (*see* Cancer).
Sero-cystic sarcoma of breast, 211
Serous membranes, tuberculosis of, 157
Shattuck, Fredk. C., arsenical poisoning with special reference to its domestic sources, 285
Shober, J. B., softening of pons Varolii, thrombosis of right internal carotid artery, 219
Sinkler, Wharton, cystic disease of liver, 69
Skin, suspected leprosy, 240
Slifer, H. F., calcified lipoma, 230
Smith, Allen J., anomaly of the kidneys, 161
case of tuberculosis treated by Koch's method, 238
kidneys from a case of gout, 160
multiple cancer involving cerebellum, 213
typhoid fever with purpura hæmorrhagica, 18
visceral syphilis, 237
Speech-centre, atrophy of motor, 220
Spinal cord in pernicious anæmia, 221
Spine, fracture of, 13
Spleen, cystic, 275
Stengel, Alfred, aortic valve disease with fatty heart, 101
cancer of liver, 50
congenital cystic kidneys, 164
cylindroids in the urine and their significance, 191
discussion, 42, 55, 56, 57, 95, 135, 139, 143, 146, 229
duodenal ulcer, 49
gall-stone causing intestinal obstruction, 68
leukæmic blood, 90
methods of examining the blood, 122
Stewart, D. D., aneurism of aorta, 81
Stomach, cancer of, 47, 48, 74, 75, 76
colloid, 37, 45

Stomach, hemorrhagic erosions of, 23
in hydrocyanic acid poisoning, 45
perforation of, by cancer, 37
in ulcer, 58
ulcer of, 58
Strongylus ovis-pulmonalis, 152
Suppuration, the factors concerned in, 248
Syphilis, visceral, 237

TAYLOR, J. M., hypospadia, dilatation of bladder, absence of left kidney, 190
Thrombosis of heart and pulmonary vessels in pneumonia, 136
of right internal carotid artery, 219
renal, in an infant, 141
Tibia, osteo-sarcoma of, 13
Tongue of cat containing Trichina spiralis, 282
Toulmin, H., croupous pneumonia and parenchymatous nephritis, 156
Trichina spiralis in cat's tongue, 282
Tubercular arthritis, 12
Tuberculosis, canine, 301
case of, treated by Koch's lymph, 238
generalized, 282
lesions secondary to pulmonary, 232
of bones and joints, 1
of gastro-intestinal tract, 235
of lungs, 157
of serous membranes, 157
of the nervous system, 232
the giant-cell of, 262
Tuberculous broncho - pneumonia, acute, 154
Typhoid fever with perforation and peritonitis, 20
with purpura hæmorrhagica, 18
Tyson, James, discussion, 186

ULCER of duodenum, 49
of stomach, 58
Ureters, bilateral double, 202
Uric acid, relations to albuminuria and Bright's disease, 169
Urine, cylindroids in the, 192
Uterus, cancer of, 166, 204, 209
fibroid of, 203, 204, 205
polypoid, 208
pregnant, with fœtus intact, 207
sarcoma of, 204

VALVULAR disease of heart, 103, 104, 134, 138
Vansant, E. L., atheroma of heart and contracted kidneys, 103
cancer of gall-bladder, 51, 73
of stomach, 47, 48
Ventricle, dilated right, of heart, 100
Vermiform appendix, cyst of, 54
Verminous pneumonia of sheep, 152
Visceral syphilis, 237
Volvulus due to extra-uterine pregnancy, 75

WESTCOTT, T. S., chronic nephritis and dilated heart, 165
Wharton, H. R., cyst of vermiform appendix, 54
dermoid cyst of mouth, 56
discussion, 13, 227
melanotic sarcoma of neck, 229
obstruction of intestines, 41
ostitis of head of femur, 17
papilloma of rectum, 56
Willard, De Forest, tuberculosis of bones and joints, 5
Wilson, W. R., congenital cystic elephantiasis, 241
Wolff, L., aneurism of aorta, 137
atheroma of heart and aorta, 136
discussion, 188
endocarditis, ulcerative, 137
hemorrhagic pericarditis, 138
Wound, gunshot, of mouth, 36

ZIEGLER, J. L., multiple embolism, 100

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